THE Journal of the Kansas Medical Society

MARCH
1958

VOL LIX
NO III

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SPECIFICALLY
for petit mal
and psychomotor seizures

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(methsuximide, Parke-Davis)

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Clinical experience[1,2,3] indicates that CELONTIN:

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1. Zimmerman, F. T., and Burgemeister, B.: *Arch. Neurol. & Psychiat.* 72:720, 1954.
2. Zimmerman, F. T., and Burgemeister, B.: J.A.M.A. 157:1194, 1955.
3. Zimmerman, F. T.: *Arch. Neurol. & Psychiat.* 76:65, 1956.

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THE JOURNAL
of the
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Volume LIX MARCH, 1958 Number 3

TABLE OF CONTENTS

Original Articles

The Medical Center: A Resume of Progress and a Look at the Future by the Dean . . . 81

Exchange Transfusions: Experiences at University of Kansas Medical Center Over a Two-Year Period—Don S. Overend, M.D., Springfield, Missouri 83

Polycythemia Vera: Study of Coagulation Abnormalities, Both Thrombotic and Hemorrhagic—Sloan J. Wilson, M.D., Helen E. Health, A.B., and William E. Larsen, M.D., Kansas City 90

Arteriovenous Fistula: Report of Case Developing after Operation for Ruptured Intervertebral Disc and Successfully Repaired by Homograft—Creighton A. Hardin, M.D., and Max Allen, M.D., Kansas City 93

Deafness: Examination Techniques for Evaluating Malingering and Psychogenic Disabilities —C. P. Goetzinger, Ph.D., and G. O. Proud, M.D., Kansas City 95

The Retarded Child: Fifty Years Ago a Custodial Problem—Now One in Education and Rehabilitation—C. Arden Miller, M.D., Kansas City 101

Primary Carcinoma of the Vagina: Results of Treatment by Radiation—Charles A. Hunter, Jr., M.D., Kansas City 106

Asian Influenza Vaccine: How Effective Was It in Prevention of Acute Respiratory Infection at the University of Kansas Medical Center—Robert A. Jordan, M.D., and Tom D. Y. Chin, M.D., Kansas City 111

Caustic Esophageal Stricture: A Review of the Literature and a Suggested Plan of Treatment —R. E. Bridwell, M.D., Kansas City . . 114

Clinicopathological Conference: Abdominal Pain, Shock, Thrombocytopenia, and Rapid Enlargement of the Liver 122

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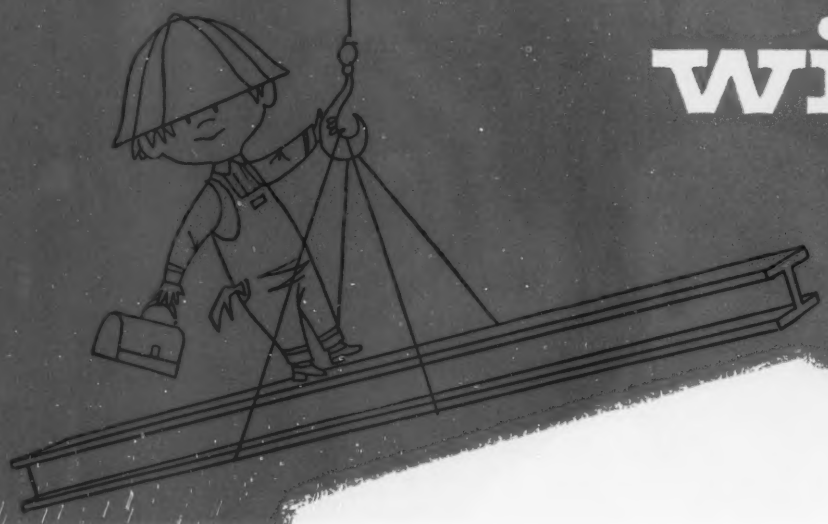

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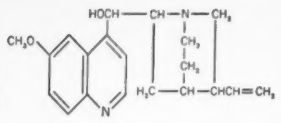

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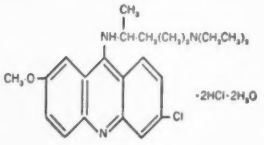

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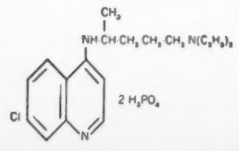

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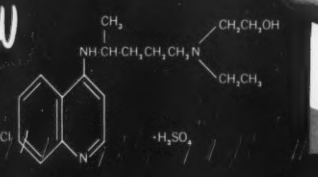

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REFERENCES:

1. Scherbel, A.L., Schuchter, S.L., and Harrison, J.W.: *Cleveland Clin. Quart.* 24:98, Apr., 1957.
2. Editorial: *Bull A. Mil. Dermatologists*, The Shock Section 5:25, Nov., 1956.
3. Cornbleet, Theodore: Arch. Dermat. 73:572, June, 1956.

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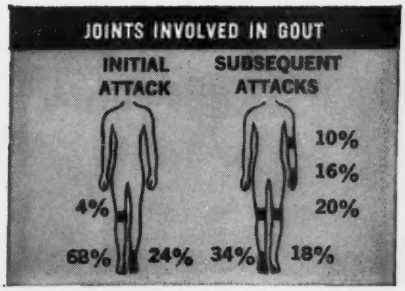

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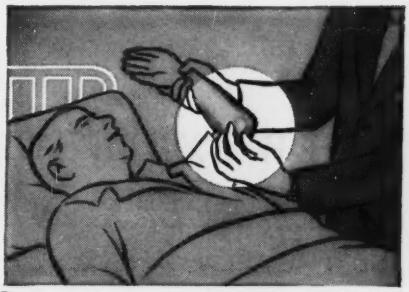
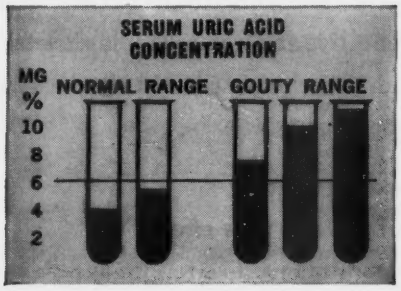
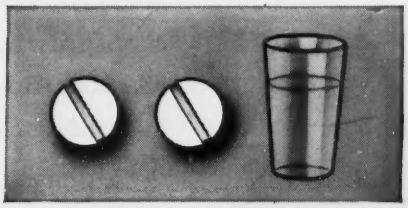


2. Enlargement of bursae such as in this case involving the olecranon bursa.



3. Elevated serum uric acid levels.



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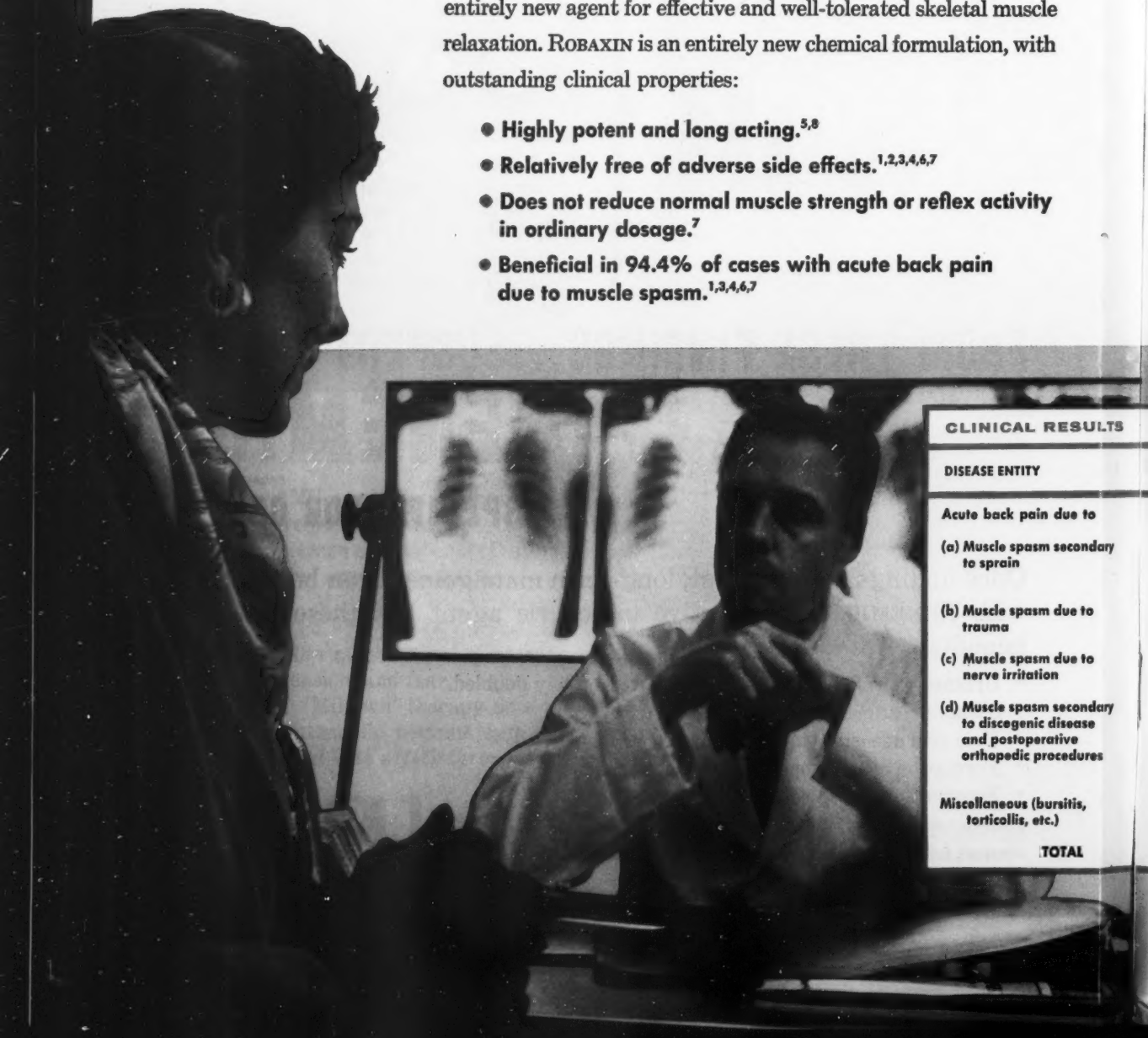
ROBAXIN – synthesized in the Robins Research Laboratories, and intensively studied for five years – introduces to the physician an entirely new agent for effective and well-tolerated skeletal muscle relaxation. ROBAXIN is an entirely new chemical formulation, with outstanding clinical properties:

- **Highly potent and long acting.**[5,8]
- **Relatively free of adverse side effects.**[1,2,3,4,6,7]
- **Does not reduce normal muscle strength or reflex activity in ordinary dosage.**[7]
- **Beneficial in 94.4% of cases with acute back pain due to muscle spasm.**[1,3,4,6,7]



CLINICAL RESULTS	WITH
DISEASE ENTITY	NO. OF CASES
Acute back pain due to	
(a) Muscle spasm secondary to sprain	18
(b) Muscle spasm due to trauma	13
(c) Muscle spasm due to nerve irritation	5
(d) Muscle spasm secondary to discogenic disease and postoperative orthopedic procedures	30
Miscellaneous (bursitis, torticollis, etc.)	6
TOTAL	72

baxin®

(Methocarbamol Robins, U.S. Pat. No. 2770649)

Highly specific action

ROBAXIN is highly specific in its action on the internuncial neurons of the spinal cord – with inherently sustained repression of multisynaptic reflexes, but with no demonstrable effect on monosynaptic reflexes. It thus is useful in the control of skeletal muscle spasm, tremor and other manifestations of hyperactivity, as well as the pain incident to spasm, without impairing strength or normal neuromuscular function.

Beneficial in 94.4% of cases tested

When tested in 72 patients with acute back pain involving muscle spasm, ROBAXIN induced marked relief in 59, moderate relief in 6, and slight relief in 3 – or an over-all beneficial effect in 94.4%.[1,3,4,6,7] No side effects occurred in 64 of the patients, and only slight side effects in 8. In studies of 129 patients, moderate or negligible side effects occurred in only 6.2%.[1,2,3,4,6,7]

WITH ROBAXIN IN ACUTE BACK PAIN[1,3,4,6,7]

NO. OF CASES	DURATION OF TREATMENT	DOSE PER DAY (divided)	RESPONSE marked	mod.	slight	neg.	SIDE EFFECTS
18	2-42 days	3-6 Gm.	17	1	0	0	None, 16 Dizziness, 1 Slight nausea, 1
13	1-42 days	2-6 Gm.	8	1	3	1	None, 12 Nervousness, 1
5	4-240 days	2.25-6 Gm.	4	1	0	0	None, 5
30	2-28 days	1.5-9 Gm.	24	3	0	3	None, 25 Dizziness, 1 Lightheadedness, 2 Nausea, 2 *
6	3-60 days	4-8 Gm.	6	0	0	0	None, 6
72			59	6	3	4	*Relieved on reduction of dose

References: 1. Carpenter, E. B.: Publication pending. 2. Carter, C. H.: Personal communication. 3. Forsyth, H. F.: Publication pending. 4. Freund, J.: Personal communication. 5. Morgan, A. M., Truitt, E. B., Jr., and Little, J. M.: American Pharm. Assn. 46:374, 1957. 6. Nachman, H. M.: Personal communication. 7. O'Doherty, D.: Publication pending. 8. Truitt, E. B., Jr., and Little, J. M.: J. Pharm. & Exper. Therap. 119:161, 1957.

Indications – Acute back pain associated with: (a) muscle spasm secondary to sprain; (b) muscle spasm due to trauma; (c) muscle spasm due to nerve irritation; (d) muscle spasm secondary to discogenic disease and postoperative orthopedic procedures; and miscellaneous conditions, such as bursitis, fibrositis, torticollis, etc.

Dosage – Adults: Two tablets 4 times daily to 3 tablets every 4 hours. Total daily dosage: 4 to 9 Gm. in divided doses.

Precautions – There are no specific contraindications to Robaxin and untoward reactions are not to be anticipated. Minor side effects such as lightheadedness, dizziness, nausea may occur rarely in patients with unusual sensitivity to drugs, but disappear on reduction of dosage. When therapy is prolonged routine white blood cell counts should be made since some decrease was noted in 3 patients out of a group of 72 who had received the drug for periods of 30 days or longer.

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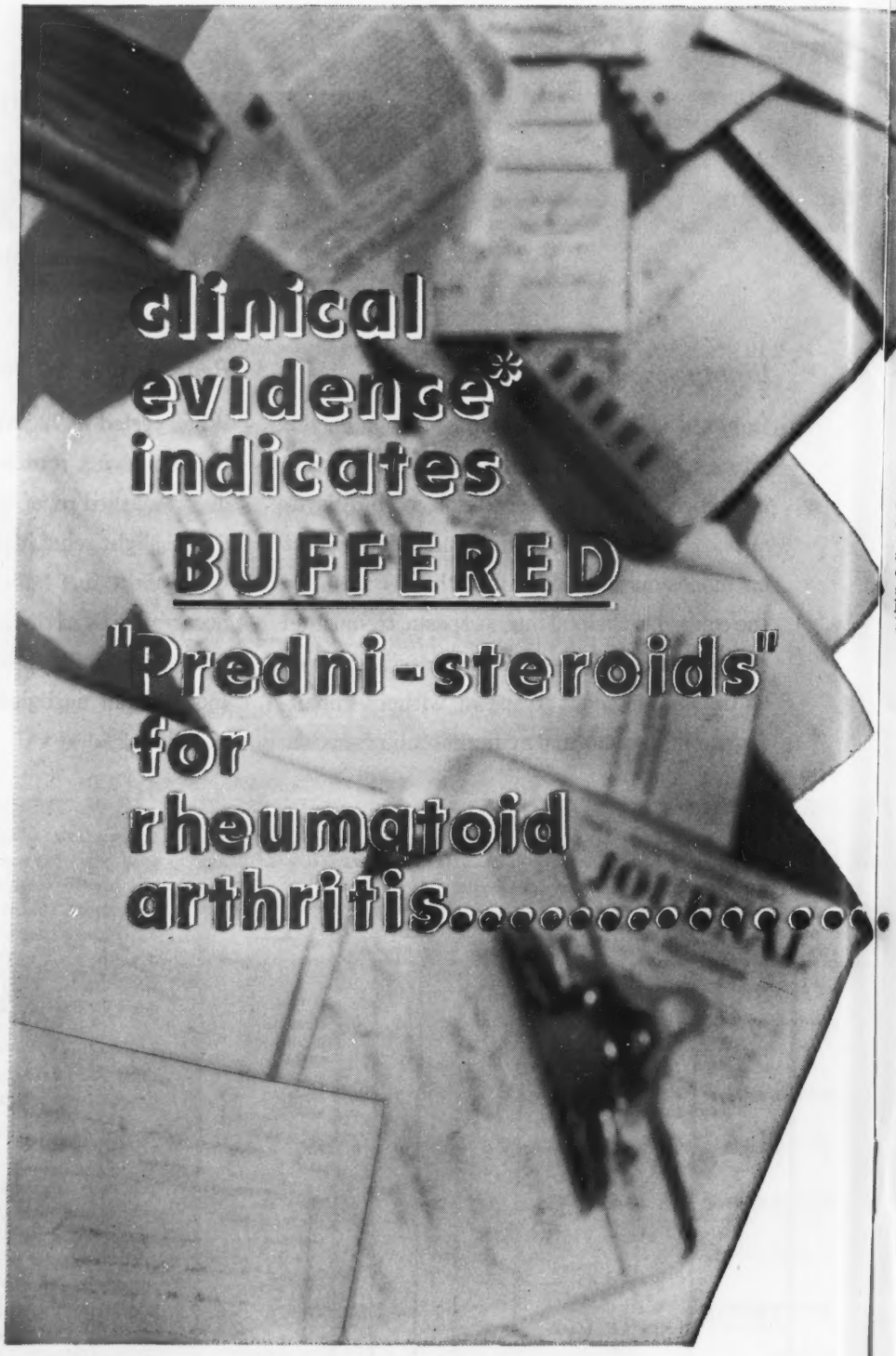

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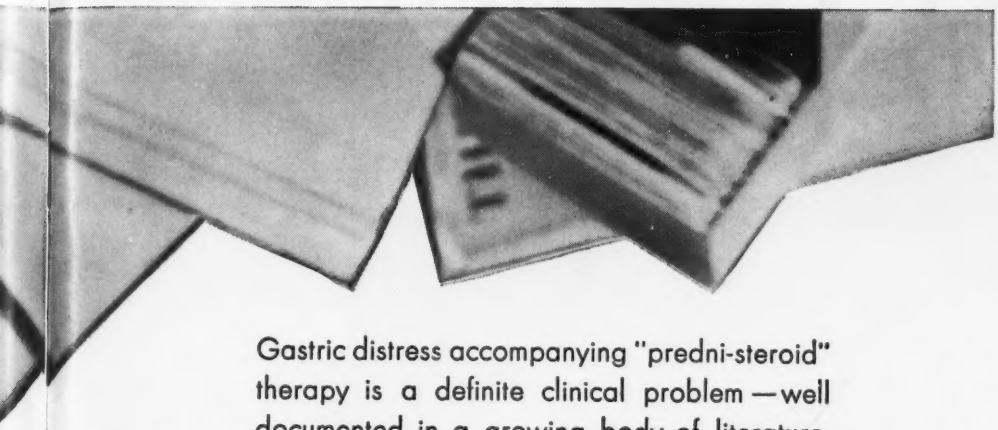

Gastric distress accompanying "predni-steroid" therapy is a definite clinical problem — well documented in a growing body of literature.

*"In view of the beneficial responses observed when antacids and bland diets were used concomitantly with prednisone and prednisolone, we feel that these measures should be employed prophylactically to offset any gastrointestinal side effects."—Dordick, J. R. *et al.*: N. Y. State J. Med. 57:2049 (June 15) 1957.

*"It is our growing conviction that all patients receiving oral steroids should take each dose after food or with adequate buffering with aluminum or magnesium hydroxide preparations."—Sigler, J. W. and Ensign, D. C.: J. Kentucky State M. A. 54:771 (Sept.) 1956.

*"The apparent high incidence of this serious [gastric] side effect in patients receiving prednisone or prednisolone suggests the advisability of routine co-administration of an aluminum hydroxide gel."—Bollet, A. J. and Bunim, J. J.: J. A. M. A. 158:459 (June 11) 1955.

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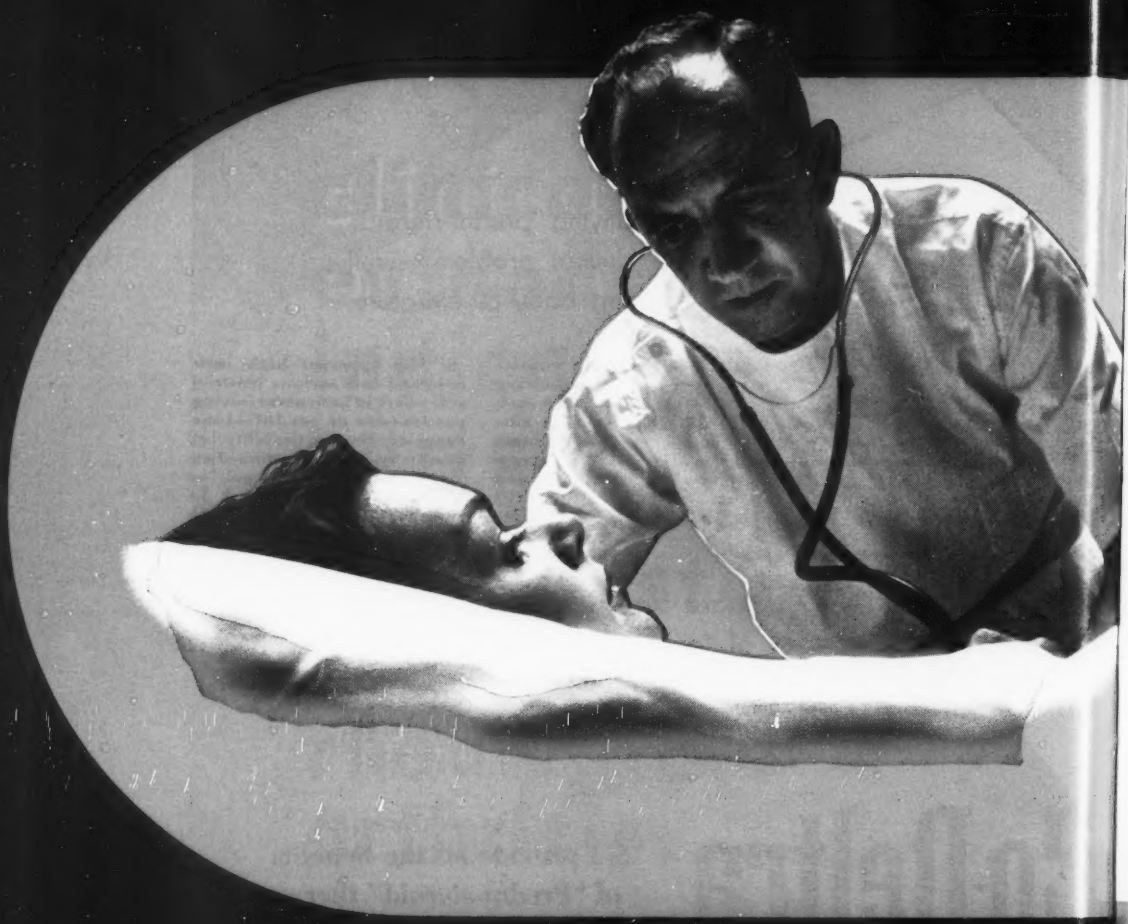

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- improves and simplifies the management of hypertension
- markedly enhances the effects of antihypertensive agents
- reduces dosage requirements for other antihypertensive agents—often below the level of distressing side effects
- smooths out blood pressure fluctuations

INDICATIONS: management of hypertension

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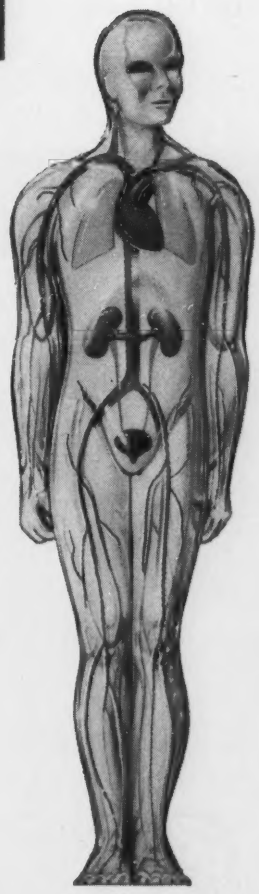

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In pain. Anxious. Fearful. On the road to cardiac invalidism. These are the pathways of angina patients. For fear and pain are inexorably linked in the angina syndrome.

For angina patients—perhaps the next one who enters your office—won't you consider new CARTRAX? This doubly effective therapy combines PETN (pentaerythritol tetranitrate) for lasting vasodilation and ATARAX for peace of mind. Thus CARTRAX relieves not only the anginal pain but reduces the concomitant anxiety.

Dosage and supplied: begin with 1 to 2 yellow CARTRAX "10" tablets (10 mg. PETN plus 10 mg. ATARAX) 3 to 4 times daily. When indicated, this may be increased for more optimal effect by switching to pink CARTRAX "20" tablets (20 mg. PETN plus 10 mg. ATARAX.) For convenience, write "CARTRAX 10" or "CARTRAX 20." In bottles of 100. CARTRAX should be taken 30 to 60 minutes *before* meals, on a continuous dosage schedule. Use PETN preparations with caution in glaucoma.

"Cardiac patients who show significant manifestations of anxiety should receive ataractic treatment as part of the therapeutic approach to the cardiac problem."[1]

1. Waldman, S., and Pelner, L.: Am. Pract. & Digest Treat. *8*:1075 (July) 1957.

*TRADEMARK

ROERIG Rx

New York 17, New York
Division, Chas. Pfizer & Co., Inc.

a new high in
anti-inflammatory effects
with lower dosage
(averages 1/3 less than
prednisone)

The
Achievements
of

Aristocort®

Triamcinolone LEDERLE

a new low in the collateral
hormonal effects associated
with all previous corticosteroids

- No sodium or water retention
- No potassium loss
- No interference with psychic equilibrium
- Low incidence of peptic ulcer and osteoporosis

Aristocort is available in 2 mg. scored tablets (pink), bottles of 30; and 4 mg. scored tablets (white), bottles of 30 and 100.

Lederle

The Achievement in Skin Diseases: In a study of 26 patients with severe dermatoses, ARISTOCORT was proved to have potent anti-inflammatory and antipruritic properties, even at a dosage only ⅔ that of prednisone.[1]... Striking affinity for skin and tremendous potency in controlling skin disease, including 50 cases of psoriasis, of which over 60% were reported as *markedly improved*[2]... absence of serious side effects specifically noted.[1,2,3]

The Achievement in Rheumatoid Arthritis: Impressive therapeutic effect in most cases of a group of 89 patients[4]...6 mg. of ARISTOCORT corresponded in effect to 10 mg. of prednisone daily (in addition, gastric ulcer which developed during prednisone therapy in 2 cases disappeared during ARISTOCORT therapy).[5]

Aristo

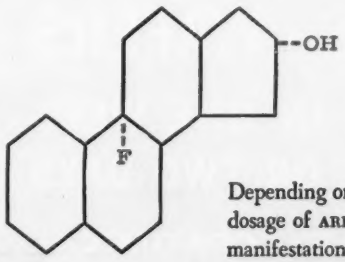
1. Rein, C. R., Fleischmajer, R., and Rosenthal, A. L.: J. A. M. A. *165*:1821, (Dec. 7) 1957.
2. Shelley, W. B., and Pillsbury, D. M.: Personal Communication.
3. Sherwood, A., and Cooke, R. A.: Personal Communication.
4. Freyberg, R. H., Berntsen, C. A., and Hellman, L.: Paper presented at International Congress on Rheumatic Diseases, Toronto, June 25, 1957.
5. Hartung, E. F.: Personal Communication.
6. Schwartz, E.: Personal Communication.
7. Sherwood, A., and Cooke, R. A.: J. Allergy 28:97, 1957.
8. Hellman, L., Zumoff, B., Kretshmer, N., and Kramer, B.: Paper presented at Nephrosis Conference, Bethesda, Md., Oct. 26, 1957.
9. Ibid.: Personal Communication.
10. Barach, A. L.: Personal Communication.
11. Segal, M. S.: Personal Communication.
12. Cooke, R. A.: Personal Communication.
13. Dubois, E. L.: Personal Communication.

The Achievement in Respiratory Allergies: "Good to excellent" results in 29 of 30 patients with chronic intractable bronchial asthma at an average daily dosage of only 7 mg.[6]... Average dosage of 6 mg. daily to control asthma and 2 to 6 mg. to control allergic rhinitis in a group of 42 patients, with an actual reduction of blood pressure in 12 of these.[7]

The Achievement in Other Conditions: Two failures, 4 partial remissions and 8 cases with complete disappearance of abnormal chemical findings lead to characterization of ARISTOCORT as possibly the most desirable steroid to date in treatment of the nephrotic syndrome.[8,9]... Prompt decrease in the cyanosis and dyspnea of pulmonary emphysema and fibrosis, with marked improvement in patients refractory to prednisone.[10,11,12]... Favorable response reported for 25 of 28 cases of disseminated lupus erythematosus.[13]

tocort®

Triamcinolone LEDERLE



Depending on the acuteness and severity of the disease under therapy, the initial dosage of ARISTOCORT is usually from 8 to 20 mg. daily. When acute manifestations have subsided, maintenance dosage is arrived at gradually, usually by reducing the total daily dosage 2 mg. every 3 days until the smallest dosage has been reached which will suppress symptoms.

Comparative studies of patients changed to ARISTOCORT from prednisone indicate a dosage of ARISTOCORT lower by about ⅓ in rheumatoid arthritis, by ⅓ in allergic rhinitis and bronchial asthma, and by ⅓ to ½ in inflammatory and allergic skin diseases. With ARISTOCORT, no precautions are necessary in regard to dietary restriction of sodium or supplementation with potassium.

ARISTOCORT is available in 2 mg. scored tablets (pink), bottles of 30; and 4 mg. scored tablets (white), bottles of 30 and 100.

LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER NEW YORK

for "This Wormy World"

Pleasant tasting

'ANTEPAR'® brand

PIPERAZINE

SYRUP · TABLETS · WAFERS

Eliminate **PINWORMS IN ONE WEEK**
ROUNDWORMS IN ONE OR TWO DAYS

PALATABLE • DEPENDABLE • ECONOMICAL

'ANTEPAR' SYRUP – Piperazine Citrate, 100 mg. per cc.
'ANTEPAR' TABLETS – Piperazine Citrate, 250 or 500 mg., scored
NEW **'ANTEPAR' WAFERS** – Piperazine Phosphate, 500 mg.

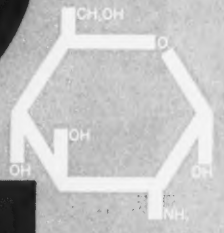
Literature available on request

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N. Y.

Three advantages of glucosamine-potentiated tetracycline:

1 ***Highest, fastest tetracycline blood levels,*** with glucosamine enhancement. A screening program involving 84 possible adjuvants, multiple four-way crossover tests, 30,000 blood level determinations and more than 100,000 assays proved glucosamine to be the enhancing agent of choice.

2 ***Greatest consistency of higher tetracycline blood levels*** Not only does glucosamine considerably increase antibiotic blood levels faster, but it produces these higher blood levels more consistently as shown by extensive crossover tests.



3 ***Achieved with the physiologic advantages of glucosamine,*** a normal human metabolite. Glucosamine, found widely in the body, is nontoxic and does not irritate the gastrointestinal tract; there is evidence that glucosamine may favorably influence the bacterial flora of the intestine. Further, it is sodium free and releases only four calories of energy per gram.

in new well-tolerated

The most widely prescribed broad-spectrum antibiotic now potentiated with glucosamine, the enhancing agent of choice

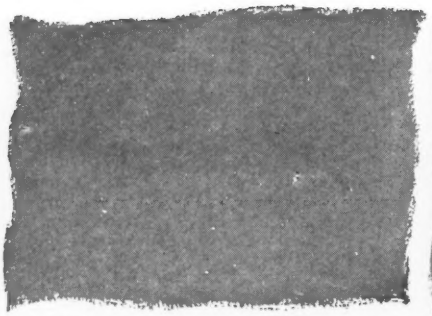
COSA-TETRACYN*

GLUCOSAMINE-POTENTIATED TETRACYCLINE

Capsules, 250 mg., 125 mg.
Half strength (125 mg. capsules) for long-term indications or pediatric use.

Pfizer Pfizer Laboratories, Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N.Y.

*Trademark



If
Monilial overgrowth
is a factor

Achrostatin* V

TETRACYCLINE (PHOSPHATE-BUFFERED) AND NYSTATIN

Combines ACHROMYCIN V with NYSTATIN

SUPPLIED:

CAPSULES contain 250 mg. tetracycline HCl equivalent (phosphate-buffered) and 250,000 units Nystatin. ORAL SUSPENSION (cherry-mint flavored) Each 5 cc. teaspoonful contains 125 mg. tetracycline HCl equivalent (phosphate-buffered) and 125,000 units Nystatin.

DOSAGE:

Basic oral dosage (6-7 mg. per lb. body weight per day) in the average adult is 4 capsules or 8 tsp. of ACHROSTATIN V per day, equivalent to 1 Gm. of ACHROMYCIN V.

ACHROSTATIN V combines ACHROMYCIN† V ...the new rapid-acting oral form of ACHROMYCIN† Tetracycline...noted for its outstanding effectiveness against more than 50 different infections ...and NYSTATIN...the antifungal specific. ACHROSTATIN V provides particularly effective therapy for those patients prone to monilial overgrowth during a protracted course of antibiotic treatment.

LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER, N. Y.
*Trademark †Reg. U. S. Pat. Off.

Lederle

Relieve moderate or severe pain

Reduce fever

Alleviate the general malaise of upper respiratory infections

'TABLOID'

'EMPIRIN' COMPOUND

WITH

CODEINE PHOSPHATE*

maximum codeine analgesia/optimum antipyretic action

*Subject to Federal Narcotic Regulations

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York

Symbols OF PROVEN PAIN RELIEF

gr. 1

gr. ½

gr. ¼

gr. ⅛

Formulas for dependable relief...

...from moderate to severe pain complicated by tension, anxiety and restlessness.

Codeine Phosphate . . . . . gr. ½
Phenobarbital . . . . . gr. ¼
Acetophenetidin . . . . . gr. 2½
Aspirin (Acetylsalicylic Acid) . . . . . gr. 3½

Codeine Phosphate . . . . . gr. ¼
Phenobarbital . . . . . gr. ¼
Acetophenetidin . . . . . gr. 2½
Aspirin (Acetylsalicylic Acid) . . . . . gr. 3½

...from pain of muscle and joint origin, simple headache, neuralgia, and the symptoms of the common cold.

'TABLOID'

'EMPIRIN' COMPOUND®

Acetophenetidin . . . . . gr. 2½
Aspirin (Acetylsalicylic Acid) . . . . . gr. 3½
Caffeine . . . . . gr. ½

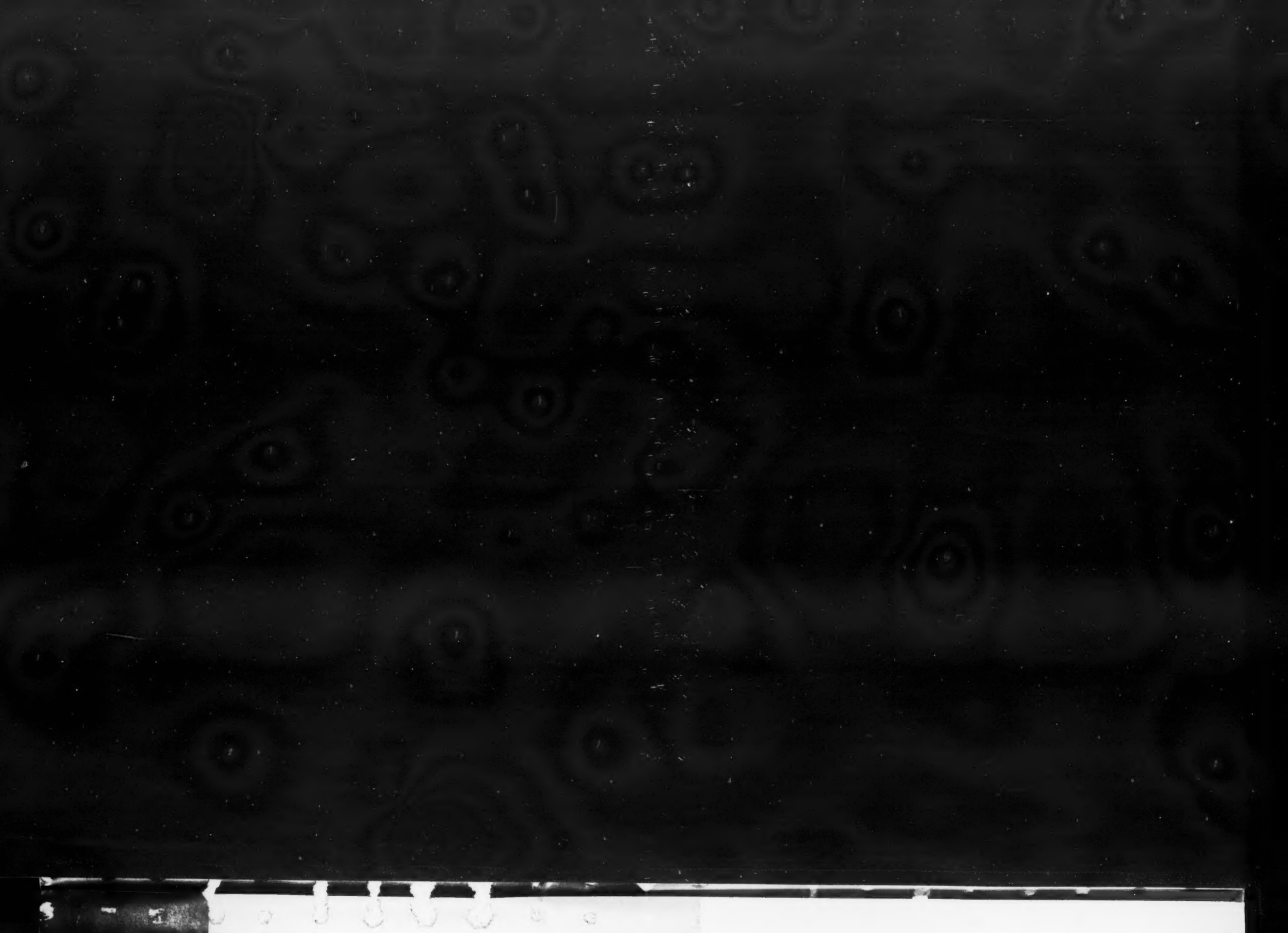
...from mild pain complicated by tension and restlessness.

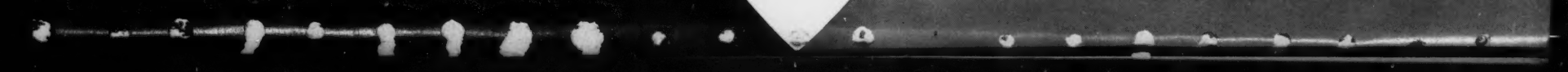
'EMPIRAL'®

Phenobarbital . . . . . gr. ¼
Acetophenetidin . . . . . gr. 2½
Aspirin (Acetylsalicylic Acid) . . . . . gr. 3½

*Subject to Federal Narcotic Regulations

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York





How Old is Too Old for Tranquilizers ?

The psychological needs of the elderly confront physicians with one of their most perplexing problems. Perhaps no other patient group suffers so much from emotional distress. Yet, precisely because of their age, geriatric patients often seem beyond the reach of tranquilizing treatment.

When tranquilization seems risky . . .

They are too much beset by complicating chronic ailments, too susceptible to serious side effects. Ataraxia is clearly indicated, yet the doctor cannot risk side reactions on liver, blood or nervous system.

Is there an answer to this dilemma?

We feel there is. In four recent papers investigators have reported good results with ATARAX in patients up to 90 years of age.* In one study, **improvement was "pronounced" in 76%, "good" in an additional 18.5%.*** ATARAX has been successfully used in such cases as senile anxiety, agitation, hyperemotivity and persecution complex.* On ATARAX, patients became ". . . quieter and more manageable. They slept better and demonstrated improved relations with other patients and hospital personnel. Even their personal hygiene improved, and they required less supervisory management."*

. . . ATARAX is safe

Yet even in the aged, ATARAX has given "no evidence of toxicity. . . . Complete liver function tests and blood studies were made on all patients after two months of therapy. . . . There were no significant abnormalities."* With still other elderly patients "tolerance to the drug was excellent, even in cases where the patients were given relatively high doses."* Similarly, no parkinsonian effects have been observed on ATARAX therapy.

Nor does ATARAX make your patients want to sleep all day. Instead, they can better take care of themselves, because ATARAX leaves them both calm and alert. In sum, ATARAX ". . . does not impair psychic function and has a minimum of side effects. . . . It appears that ATARAX is a safe drug. . . ."*

These, undoubtedly, are the results you want when emotional problems beset your geriatric patients. For the next four weeks, won't you prescribe tiny ATARAX tablets or pleasant-tasting ATARAX syrup — both so readily acceptable to the elderly.

*Documentation on request

ATARAX®
(BRAND OF HYDROXYZINE)

M. A. Sidell M.D.
Medical Director

ROERIG
New York 17, New York
Division, Chas. Pfizer & Co., Inc.

ATARAX
in any hyperemotive state

for childhood behavior disorders
10 mg. tablets—3-6 years, one tablet t.i.d.; over 6 years, two tablets t.i.d. Syrup—3-6 years, one tsp. t.i.d.; over 6 years, two tsp. t.i.d.

for adult tension and anxiety
25 mg. tablets one tablet q.i.d. Syrup—one tbsp. q.i.d.

for severe emotional disturbances
100 mg. tablets—one tablet t.i.d.

for adult psychiatric and emotional emergencies
Parenteral Solution—25-50 mg. (1-2 cc.) intramuscularly, 3-4 times daily, at 4-hour intervals. Dosage for children under 12 not established.

Supplied: Tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

WHEN YOU TREAT INFECTIONS IN PATIENTS SUCH AS THESE

- debilitated
- elderly
- diabetics
- infants, especially prematures
- those on corticoids
- those who developed moniliasis on previous broad-spectrum therapy
- those on prolonged and/or high antibiotic dosage
- women—especially if pregnant or diabetic

the best broad-spectrum antibiotic to use is

MYSTECLIN-V

Squibb Tetracycline Phosphate Complex (Sumycin) and Nystatin (Mycostatin) — Sumycin plus Mycostatin

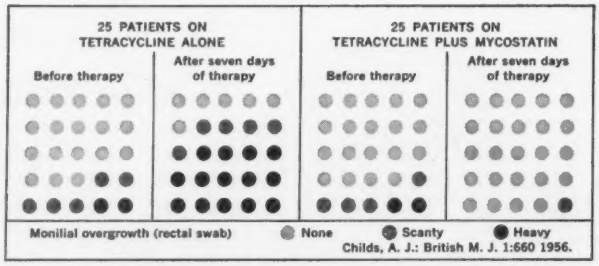
for practical purposes, Mysteclin-V is sodium-free

for "built-in" safety, Mysteclin-V combines:

1. Tetracycline phosphate complex (Sumycin) for superior initial tetracycline blood levels, assuring fast transport of adequate tetracycline to the infection site.

2. Mycostatin—the first safe antifungal antibiotic—for its specific antimonilial activity. Mycostatin protects many patients (see above) who are particularly prone to monilial complications when on broad-spectrum therapy.

MYSTECLIN-V PREVENTS MONILIAL OVERGROWTH



Childs, A. J.: British M. J. 1:660 1956.

Capsules (250 mg./250,000 u.), bottles of 16 and 100. *Half-Strength Capsules* (125 mg./125,000 u.), bottles of 16 and 100. *Suspension* (125 mg./125,000 u.), 2 oz. bottles. *Pediatric Drops* (100 mg./100,000 u.), 10 cc. dropper bottles.

SQUIBB

Squibb Quality—the Priceless Ingredient

"MYSTECLIN," "MYCOSTATIN," AND "SUMYCIN" ARE SQUIBB TRADEMARKS

SEARLE *announces...*

a superior psychochemical
for the management of both
minor and major
emotional disturbances

Dartal T.M.

dihydrochloride brand of thiopropazate dihydrochloride

- more effective than most potent tranquilizers
- as well tolerated as the milder agents
- consistent in effects as few tranquilizers are

Dartal is a unique development of Searle Research, proved under everyday conditions of office practice

It is a single chemical substance, thoroughly tested and found particularly suited in the management of a wide range of conditions including psychotic, psychoneurotic and psychosomatic disturbances.

Dartal is useful whenever the physician wants to ameliorate psychic agitation, whether it is basic or secondary to a systemic condition.

In extensive clinical trial Dartal caused no dangerous toxic reactions. Drowsiness and dizziness were the principal side effects reported by non-psychotic patients, but in almost all instances these were mild and caused no problem.

Specifically, the usefulness of Dartal has been established in psychoneuroses with emotional hyperactivity, in diseases with strong psychic overtones such as ulcerative colitis, peptic ulcer and in certain frank and senile psychoses.

Usual Dosage • In psychoneuroses with anxiety and tension states *one 5 mg. tablet t.i.d.*

• In psychotic conditions *one 10 mg. tablet t.i.d.*

RELIEF

1

2

FROM INFECTION*

FROM IRRITATION

*as adjunctive therapy only

THE FIRST TROCHE TO PROVIDE THREEFOLD BENEFITS

'PENTAZETS'

TROCHES

NON-NARCOTIC ANTITUSSIVE EFFICACY SHOWN TO APPROXIMATE THAT OF CODEINE

AND **NOW** COUGH CONTROL TOO

With the addition of a non-narcotic antitussive to troche medication, 'PENTAZETS' provides a new and extended therapeutic advantage in this convenient form of treatment.

Treatment of the cough too, so often a troublesome symptom of sore throat, combined with wide-range antibiotic activity and soothing analgesic benefit, now offers *three*fold relief in a variety of throat irritations.

And 'PENTAZETS' are pleasant-tasting, too, making them highly acceptable, especially to children.

'PENTAZETS' contains:

• *Homarylamine*—a new non-narcotic antitussive with cough control shown to approximate that of codeine. • *Bacitracin-Tyrothricin-Neomycin*—a combined antibiotic treatment against many pathogenic organisms with little danger of unfavorable side effects. • *Benzocaine*—a local anesthetic for soothing relief to inflamed tissues. Being slowly absorbed, it is especially beneficial for prolonged effect and benefit to surrounding areas.

Supplied: Vials of 12.

Each 'PENTAZETS' troche contains:	
Homarylamine hydrochloride	20 mg.
Zinc Bacitracin	50 units
Tyrothricin	1 mg.
Neomycin sulfate (equivalent to 3.5 mg. neomycin base)	5 mg.
Benzocaine	5 mg.

MSD

MERCK SHARP & DOHME
DIVISION OF MERCK & CO., INC., PHILADELPHIA 1, PA.

PENTAZETS is a trademark of Merck & Co., Inc.

ACHROCIDIN*

TETRACYCLINE-ANTIHISTAMINE-ANALGESIC COMPOUND LEDERLE

A versatile, well-balanced formula offering in one tablet the drugs often prescribed separately for treating upper respiratory infections.

Traditional and nonspecific nasopharyngeal symptoms of malaise and chilly sensations are rapidly relieved, and headache, muscular pain, and pharyngeal and nasal discharges are reduced or eliminated.

Early effective therapy is provided against such bacterial complications as sinusitis, otitis, bronchitis and pneumonitis to which the patient may be highly vulnerable at this time.

Adult dosage for ACHROCIDIN Tablets and new, caffeine-free ACHROCIDIN Syrup is two tablets or teaspoonfuls of syrup three or four times daily. Dosage for children reduced according to weight and age.

Available on prescription only.

TABLETS *(Sugar-coated)*
Each tablet contains:

ACHROMYCIN® Tetracycline	125 mg.
Phenacetin	120 mg.
Caffeine	30 mg.
Salicylamide	150 mg.
Chlorothen Citrate	25 mg.

Bottles of 24 and 100

SYRUP *(Lemon-lime flavored)*
Each teaspoonful (5 cc.) contains:

ACHROMYCIN® Tetracycline equivalent to tetracycline HCl	125 mg.
Phenacetin	120 mg.
Salicylamide	150 mg.
Ascorbic Acid (C)	25 mg.
Pyrilamine Maleate	15 mg.
Methylparaben	4 mg.
Propylparaben	1 mg.

Bottle of 4 oz.

LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER, NEW YORK
*Trademark

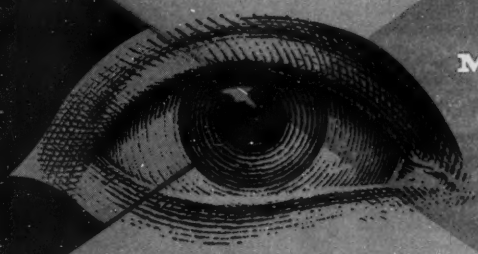
Lederle

for simultaneously combating inflammation, allergy, infection

METIMYD® ophthalmic suspension
(0.5% prednisolone acetate and 10% sulfacetamide sodium—5 cc. dropper bottle)

METIMYD ointment with neomycin
(0.5% prednisolone acetate, 10% sulfacetamide sodium and 0.25% neomycin sulfate—⅛ oz. tube)

in external eye disorders look to these topical healing aids



for ocular allergies

METRETON®
ophthalmic suspension
(0.2% prednisolone acetate and 0.3% CHLOR-TRIMETON®—5 cc. dropper bottle)

standard for ocular infections

Sodium SULAMYD® ophthalmic solution 30%
(Sulfacetamide Sodium U.S.P.—5 and 15 cc. dropper bottles)

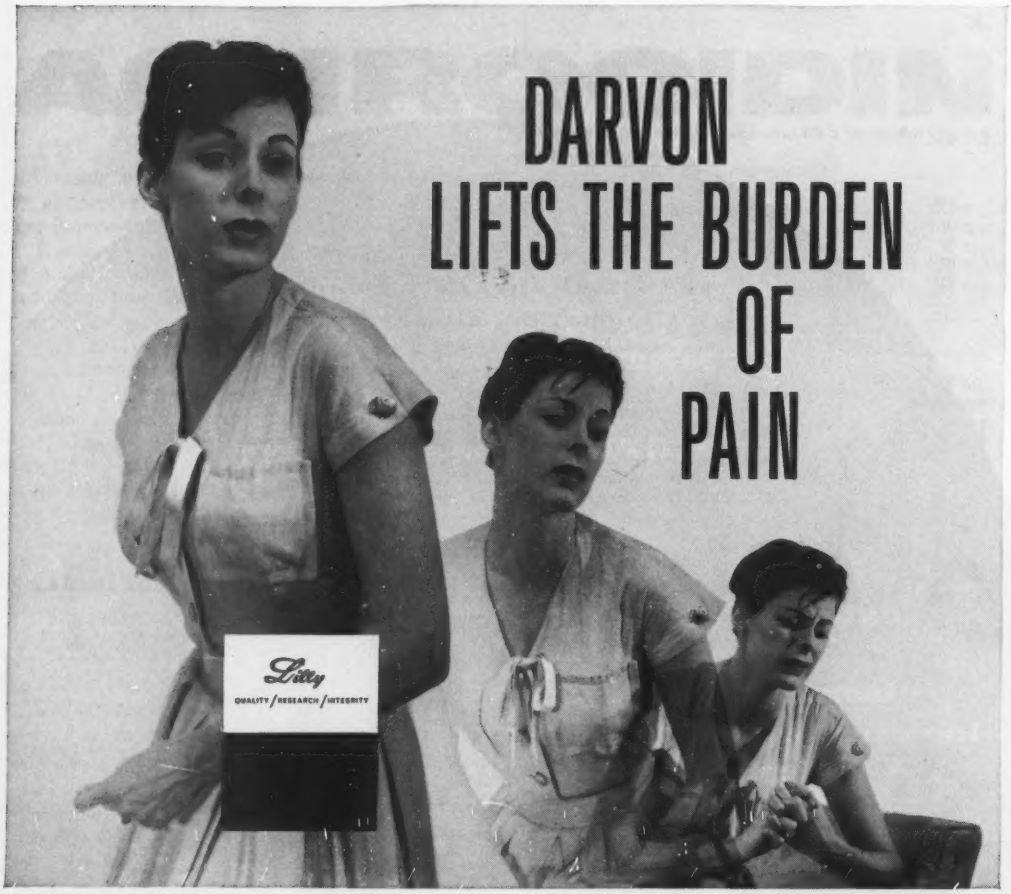
Sodium SULAMYD ophthalmic solution 10% with Methylcellulose 0.5% (15 cc. dropper bottle)

Sodium SULAMYD ophthalmic ointment 10%
(⅛ oz. tube)

SCHERING CORPORATION • BLOOMFIELD, NEW JERSEY

Schering

M-J-128



The non-narcotic analgesic with the potency of codeine

DARVON (Dextro Propoxyphene Hydrochloride, Lilly) is equally as potent as codeine yet is much better tolerated. Side-effects, such as nausea or constipation, are minimal. You will find 'Darvon' helpful in any condition associated with pain. The usual adult dose is 32 mg. every four hours or 65 mg. every six hours as needed. Available in 32 and 65-mg. pulvules.

DARVON COMPOUND (Dextro Propoxyphene and Acetylsalicylic Acid Compound, Lilly) combines the antipyretic and anti-inflammatory benefits of 'A.S.A. Compound'* with the analgesic properties of 'Darvon.' Thus, it is useful in relieving pain associated with recurrent or chronic disease, such as neuralgia, neuritis, or arthritis, as well as acute pain of traumatic origin. The usual adult dose is 1 or 2 pulvules every six hours as needed.

Each Pulvule 'Darvon Compound' provides:

'Darvon'	*32 mg.*
Acetophenetidin	*162 mg.*
'A.S.A.' (Acetylsalicylic Acid, Lilly)	*227 mg.*
Caffeine	*32.4 mg.*

*'A.S.A. Compound' (Acetylsalicylic Acid and Acetophenetidin Compound, Lilly)

ELI LILLY AND COMPANY • INDIANAPOLIS 6, INDIANA, U. S. A.

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The JOURNAL *of the*
KANSAS MEDICAL SOCIETY

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Volume LIX **MARCH, 1958** **No. 3**

The Medical Center

A Resume of Progress and a Look at the Future by the Dean

It is a pleasure once more to be able to write a prefatory statement for the Medical Center issue of the JOURNAL OF THE KANSAS MEDICAL SOCIETY. The year just past proved to be an active one, and the year immediately ahead promises to be one of continued growth and progress. The following are some statements of fact relating to the state of the Medical Center.

Personnel. The past year saw changes in faculty, both loss and gain. On January 1, 1957, Dr. Leonard Peltier joined us as professor and head of the Section of Orthopedic Surgery. Dr. Peltier came to us from the University of Minnesota, where he had been acting head of orthopedic surgery. On July 1, Dr. L. R. C. Agnew took up his appointment as associate professor of the history of medicine and chairman of the Department of the History of Medicine. Dr. Agnew is a medical graduate of Glasgow University and a diplomate of the American Board of Pathology. He came to us from Harvard, where he obtained his master's degree in the history of science. We are extremely fortunate to have him, a physician, scholar, and professional historian, to continue the traditions of the department ably headed in the past by Dr. Logan Clendening and Dr. Ralph Major.

With a great sense of loss, we report the resignations of Dr. Harlin Firminger, Dr. Chauncey Bly, Dr. Frank Mantz, and Dr. Perry Morgan. On July 1, Dr. Firminger, a member of our faculty for six years, resigned to become professor of pathology and chairman of the Department of Pathology in the University of Maryland School of Medicine. At the same time Dr. Mantz entered the private practice of pathology in Kansas City, Missouri, and Dr. Bly took up his duties in pathology in Rochester, New York, from which place he had joined us six years ago. On January 1, Dr. Perry Morgan resigned his position as associate professor of microbiology to become director of the Community Blood Bank of Kansas City.

At present Dr. Kurt Reissmann is serving as visiting professor of physiology in the University of the Philippines under the sponsorship of the China Medical Board. This appointment does great honor to Dr. Reissmann and to the University of Kansas as well.

On June 1, Mr. Robert Molgren, hospital administrator for five years, resigned to take up duties in a similar capacity at St. Luke's Hospital, Kansas City, Missouri. Mr. Molgren served in the most trying days of the center, in the midst of its greatest expansion. Capably he stabilized its budget and its personnel. The center owes him a tremendous debt of gratitude.

Coincident with his leaving, the organizational pattern of the center was revised. Similar positions of associate dean and associate director were created, the former occupied by Dr. Vernon Wilson and the latter by Mr. Russell Miller. Mr. Miller is probably known to most of you. He is a native of Iowa, a graduate of Central College, and has his master's degree in hospital administration from the University of Minnesota. Because he has been with us in an administrative capacity for four years and has gained the confidence and affection of all our personnel, the transition in administration was acccomplished smoothly.

Dr. Edward H. Hashinger. At the time of the appearance of this issue, Dr. Edward H. Hashinger will have retired from his faculty position as professor of medicine and gerontology and head of the latter department. I know of no one who has given so unstintingly of his time, energy, and talents for the university and for medicine. He has been a stalwart for this medical school for 37 years. He has passed through all its faculty ranks, he has served as commanding officer of its military unit, he has served as

its acting dean; in short, he has done everything. He and his lovely lady have both earned our Medal of Honor.

Facilities. In September the long-needed library unit of the Medical Center was dedicated formally. This magnificent addition (we believe it to be one of the finest of its kind anywhere) paves the way for the ultimate consolidation of the medical school on the Kansas City campus. By action of the Board of Regents, the library is now named permanently the *Clendening Medical Library,* in honor and memory of Dr. Logan Clendening, physician, teacher, scholar, bibliophile, and litterateur.

In December, the new addition for Postgraduate Medical Education was completed and occupied. This unit provides centralized offices for the department immediately proximate to the scene of its major activities, Battenfeld Auditorium. Your postgraduate activities will be enhanced greatly by this addition.

On January 29, Kansas Day, the deed to the Maccochaque School property, just south of Olathe Boulevard, was recorded in Wyandotte County Courthouse in the name of the state of Kansas. Thus culminated a five-year effort. Funds for its acquisition were provided in the legislative session of 1957; the appropriation, to provide vitally needed land for expansion of the campus, represented the first major land purchase of the state for the medical school campus.

On May 25 we plan to dedicate the Children's Rehabilitation Unit. This facility was made possible by the joint efforts of many, primary among them the Society of 40 and 8 of Kansas and the Kansas Society for Crippled Children. The donors of this building are legion; we are deeply grateful to them all.

By the time this appears, the connecting tunnel between the Psychiatry Building and the Student Center-Continuation Study Building will have been completed. This vital link will increase enormously the utilization of Battenfeld Auditorium for teaching purposes.

The planning appropriation for our building to house the departments of anatomy, physiology, and biochemistry is now in hand. Hopefully, next year's Medical Center issue will contain its preliminary plans.

Lastly, I would mention a significant gift of property. In November, Mr. and Mrs. Kenneth A. Spencer, loyal and devoted alumni of the university, gave to the Kansas University Endowment Association their lovely home situated on two acres of land in Mission Hills, the same to be used as the official residence for the dean of the School of Medicine. For this magnificent gift, I am officially and personally grateful. In it, our alumni and Kansas medical colleagues will always be welcome.

Budget. All of us are proud to report that the finances of the Medical Center are in balance. Next year's budget will be approximately eight million dollars, of which five will be represented by earned income, grants, and gifts. The three million dollars represented by legislative appropriations is exactly what was requested. Our presentations were accepted and dealt with generously by the governor and the legislature. We intend that their expression of confidence will be returned with interest in further growth in stature and quality of our program.

I regret that limitations of space do not permit more extensive comments about these matters. The success of this school depends upon many factors: the adequacy of its facilities, the capacity of its students, and the excellence of its faculty. We are proud of all three. In particular, we are proud of the enthusiasm and energy of the faculty displayed in our postgraduate effort, under the one whom we consider to be the dean of all postgraduate educators, Dr. Mahlon Delp.

The relationships enjoyed by this medical school with the Kansas Medical Society are, I believe, the best enjoyed by any school in this country with its society. For all of the help, the support, the encouragement, and the interest of the Society we are extremely grateful.

All of us trust that this issue will provide something of interest for each of you. Your comments and suggestions will be appreciated.

W. Clarke Wescoe, M.D.

There is no reason to make either books or education easy, any more than tennis or football is easy. . . . Books require a certain amount of hard work and practice and, like sports, they can be both a challenge and a delight.

—Gilbert W. Chapman

Exchange Transfusions

Experiences at University of Kansas Medical Center Over a Two-Year Period

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The value of exchange transfusion in hemolytic disease of the newborn has been convincingly demonstrated since the introduction of this procedure in the early 1940's. Exchange transfusion is performed to prevent kernicterus and the brain damage which occurs in its presence. Kernicterus is associated with high serum levels of indirect bilirubin. Waters and coworkers[1] have demonstrated by spectrophotometric and biochemical studies that the yellow pigment staining the tissues of the brain stem in autopsied cases of kernicterus is bilirubin. The in vitro studies of Day[2] have shown that indirect bilirubin is a protoplasmic poison. Indirect bilirubin prepared commercially depresses the oxygen uptake of various tissues; oxidation of the commercial bilirubin or the addition of cytochrome C to the preparation renders it non-toxic. In the body, indirect bilirubin is detoxified by conjugation with glucuronic acid and subsequent conversion to direct bilirubin. Direct-reacting bilirubin has not been shown to have any toxic effect.

When indirect bilirubin accumulates in the body of the newborn infant, kernicterus is a threat. While there is a statistical correlation between high levels of serum indirect bilirubin and kernicterus, the exact level at which this occurs is not known. Hsia and others[3] have shown that the risk of kernicterus is greater as the indirect bilirubin level exceeds 20 mgm./100 ml. This is an arbitrary figure and may be subject to revision as more cases are studied. Some workers feel that in premature infants the bilirubin concentration at which kernicterus is statistically uncommon is considerably lower than 20 mgm./100 ml.

There are a number of conditions which may cause hyperbilirubinemia in the newborn period. Hemolytic disease of the newborn due to incompatibilities in the Rh, ABO, and other blood-group systems is a well-known entity and needs no further discussion. The reader desiring authoritative information on hemolytic disease of the newborn is referred to the recent review articles by Allen and Diamond,[7] Neel,[8] and Wheeler and Ambuel.[9] Sepsis occurring in the newborn period may cause elevation of the indirect bilirubin. The septic process may be due to a bacterial invader, congenital syphilis, toxoplasmosis, or cytomegalic inclusion body disease. Any of these conditions can give a clinical picture indistinguishable from erythroblastosis due to blood-group incompatibility. Each may be diagnosed by appropriate laboratory studies.

Premature infants, particularly those with respiratory difficulty, tend to have high levels of indirect bilirubin in the absence of demonstrable blood-group incompatibility or of sepsis. C. A. Miller and Reed[10] have shown that premature infants in respiratory Group III of Miller[11] more frequently develop hyperbilirubinemia which is not associated with blood incompatibility or sepsis than do term infants and premature infants in respiratory Groups I and II.

Indications for this life-saving procedure are reviewed and the technique utilized at the University of Kansas Medical Center is described.

Whatever the cause of the hyperbilirubinemia, when indirect bilirubin reaches a concentration which threatens kernicterus, a prompt adequate exchange transfusion is the only available means of preventing damage to the brain from kernicterus.

The technique of exchange transfusion as practiced at the University of Kansas Medical Center has been developed from procedures employed at many medical centers with some modifications made by personnel here. It is presented in some detail for the reader who may desire a point of reference.

The Exchange Transfusion Team. This team consists of the transfusionist, an assistant physician, a nurse, and a recorder. An operating room is the location of choice because of the need for aseptic technique and also for easy availability of special equipment.

Blood. The amount of blood to be used in the exchange procedure is determined as being two times the circulating blood volume, which is approximately 16 per cent of the body weight in kilograms. The blood chosen should be fresh (maximum time in storage, 24 hours) to avoid hemolysis and high levels of potassium. No distinction is made between blood

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from male and female donors. The blood is cross-matched by standard techniques. If the mother and infant are of the same ABO blood group, group-specific blood is chosen, otherwise group O blood is used. Rh negative blood is chosen if either the mother or the infant is Rh negative; otherwise, Rh positive blood is employed. Appropriate A or B group-specific substance is added before type O blood is given to a type A or B infant. The donor unit is cross-matched with maternal blood if possible. If maternal blood is not available, cross-matching is against the cord blood (second choice) or the blood of the infant (third choice).

In exchange transfusions of infants with jaundice complicated by anemia, the donor blood is concentrated by withdrawal of 50 to 100 cc. of plasma from the 500 cc. unit.

The blood is warmed to approximate room temperature immediately before use by immersion of the blood container in tepid water. Fenwal plastic blood containers (Fenwal Laboratories, Framingham, Massachusetts) are used at the University of Kansas Medical Center, and these require only 10 to 20 minutes of warming.

Preparation of the Patient. The infant is transported in a heated crib. Oral intake is withheld at least 4 hours prior to exchange or, if this is not possible, the stomach is emptied by gastric tube. The infant is restrained on a circumcision board and is well covered to prevent loss of body heat. Preparation of the sterile operative field is by usual techniques.

Cannulation. Those infants exchanged within the first 24 hours of life can usually be cannulated via the umbilical vein. The umbilical stump will remain in operable condition for 48 to 72 hours if kept in wet (saline) packs, and this procedure is started at birth in those infants for whom the possibility of later exchange exists.

Those infants transfused after the cord is no longer usable are cannulated via the greater saphenous or common femoral vein. The saphenous vein is exposed by a femoral cutdown, and if this structure is too small to be cannulated the femoral vein is used as second choice. The K-32 premature infant feeding tube (Pharmaseal Laboratories, Glendale, California) has been found to be most satisfactory for umbilical and femoral cannulation. This tube is furnished unsterile and is sterilized by immersion for one hour in benzalkonium chloride solution, 1:1000. The cannula is inserted well into the vessel (6 to 10 cm. in the cord and 10 to 15 cm. in femoral cutdowns) to assure its location in the vena cava. The cannula is secured into the vein by a firmly placed tie.

The Transfusion Set. We have somewhat simplified the technique of exchange transfusion by eliminating as much as possible all wettable surfaces and areas of constriction and turbulence from the transfusion pathway. In the past, considerable clotting was encountered in three-way stopcocks and in needle hubs in the pathway. Needle hubs have been eliminated by the use of the all plastic feeding tube, and the need for three-way stopcocks has been eliminated by use of the Fenwal pressure-metering recipient set (HB282, Fenwal Laboratories, Framingham, Massachusetts). This unit provides a continuous non-wettable pathway from the blood container to the vein. Figure 1 illustrates the complete transfusion pathway. The outside surfaces of the recipient tubing are sterilized by immersion in benzalkonium chloride solution (1:1000). Syringes are washed after each filling in physiological saline containing 1.0 ml. heparin sodium solution per 1000 ml. saline.

Procedure. Blood is withdrawn and administered in 20 ml. increments. Very ill or premature infants are exchanged by 10 ml. increments. Withdrawal is usually done first, and the first specimen is retained for the determination of the pre-transfusion bilirubin concentration. Twenty (or 10) ml. are withdrawn, the syringe is changed, and a similar amount is administered. This is repeated until the entire amount of the exchange is completed. At each withdrawal or administration the recorder notes the time, amount, and running total.

At the completion of each 100 ml., 1.0 to 1.5 ml. of 10 per cent calcium gluconate is administered through the cannula. In very small infants, 1.5 ml. of calcium gluconate is given at each 50 ml. of exchange. Calcium gluconate is given cautiously with the assistant checking the apical pulse. Marked bradycardia is indication for slowing or discontinuing the calcium gluconate.

The transfusion procedure is done at a rate to occupy at least one hour and preferably 1½ to 2 hours from the first withdrawal of blood to the final administration or withdrawal. "Topping-off," the administration of a final additional amount of blood over the amount of the exchange, is not done at this

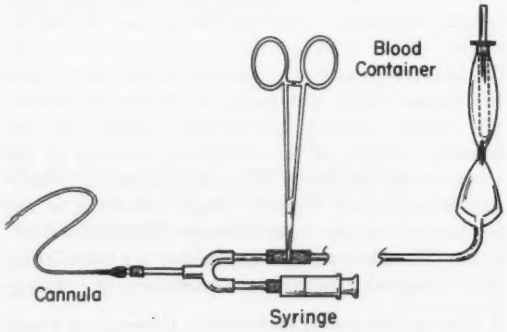

Figure 1

hospital. A portion of the final withdrawal specimen is retained for a determination of the post-transfusion bilirubin concentration.

Throughout the procedure the infant is carefully observed for cyanosis, for excessive irritability which may indicate impending tetany, and for vomiting. Oxygen by face mask is administered throughout the procedure in an attempt to assist the infant in oxygenating the desaturated donor blood. A suction machine and airways are always available. At the conclusion of the procedure the cannula is withdrawn and the operated vein is ligated.The skin is closed by usual surgical techniques.

Post-Transfusion Care. The infant is returned to the nursery immediately after exchange transfusion. The infant remains in a heated crib for two to four hours after transfusion, and feedings are withheld for four to eight hours, depending on the condition of the infant. Antibiotics are routinely given for 48 hours post-transfusion. Intramuscular procaine penicillin, 50,000 units, and streptomycin, 50 mg., are given twice daily unless some other antibiotic is indicated.

The concentrations of hemoglobin and bilirubin are checked the morning after exchange transfusion, and bilirubin determinations are continued until a definite downward trend is seen. The concentration of bilirubin 12 to 18 hours after exchange is usually somewhat higher than that immediately post-exchange, presumably due to the liberation of tissue bilirubin. If the bilirubin continues to rise after the first exchange transfusion, a second exchange transfusion is done when the indirect bilirubin again approaches or reaches 20 mgm./100 ml.

Dangers of Exchange Transfusion. Hypocalcemic tetany with convulsions, hypoxia, circulatory collapse, or cardiac arrest may occur during exchange transfusion. These most commonly occur in severely jaundiced or frankly kernicteric infants. In the series to be reported here, only one infant expired during transfusion. This infant had clinical signs of kernicterus prior to exchange. One other infant showed tetany during exchange which responded promptly to administration of additional calcium gluconate.

Results of Exchange Transfusion

During the period of two years from July 1, 1955, to June 31, 1957, 46 exchange transfusions were performed at the University of Kansas Medical Center on 43 patients. The source, race, sex, and birth weights of these patients are recorded in Table 1. This table reveals that there is a comparable number of male and female infants. There is a preponderance of white patients, although the Department of Obstetrics at this hospital reports the ratio of Negro to white births to be 6:4. Seven of 43 patients are premature, the remaining 36 weighing more than 2,500 gm. at birth. Nine of the patients reported here were referred from other hospitals, and the remainder were born at this institution. One additional exchange transfusion was performed on an infant with hemolytic phenomena and severe jaundice secondary to a large cutaneous cavernous hemangioma. This case has been excluded from the reported series.

The conditions prompting exchange transfusion included hemolytic disease of the newborn due to feto-maternal incompatibility of the Rh factor, or of the ABO blood groups, and in some cases without demonstrable incompatibility, the latter of which will be referred to hereinafter as idiopathic hyperbilirubinemia. Hemolytic disease due to Hr, Kell, or Duffy factors and sepsis were sought for but were not encountered. Rh incompatibility was demonstrable in 19 patients (44 per cent), ABO incompatibility in 12 patients (28 per cent) and idiopathic hyperbilirubinemia in 12 patients (28 per cent). A second exchange transfusion was required in three patients because of recurrence of high bilirubin concentrations. These three patients were all erythroblastotic because of incompatibility of the Rh factor.

Table 2 summarizes the results of exchange transfusion reported here. Four patients in the series expired; three of these were referred late from other hospitals when evidence of brain damage was already present. The fourth fatality occurred at 52 hours of age after exchange transfusion at six hours of age; death was the result of hyaline membrane disease complicated by marked emphysema and pneumothorax.

Table 1.

Distribution of patients* receiving exchange transfusions at University of Kansas Medical Center, July 1, 1955 to June 30, 1957.

		No. of pts.	% of pts.
Sex	Male	21	49
	Female	22	51
Race	White	27	63
	Negro	16	37
Birth Weight	2500 Gm.	36	84
	2500 Gm.	7	16
Source	Born KUMC	34	79
	Referred	9	21

*Total exchange transfusions 46; total number patients 43.

Table 2.

Results of exchange transfusions performed on 43 patients; all types of hyperbilirubinemia.

	No. of pts.	% of pts.
Satisfactory result	37	86
Unsatisfactory result	6	14
Survived with brain damage	2	
Expired	4	
Kernicterus	3	
Hyaline membranes	1	

Only one of the four deaths occurred during the exchange procedure.

Two patients in the series with clinical evidence of kernicterus have survived. One of these had idiopathic hyperbilirubinemia and is known to be brain-damaged. The other infant had erythroblastosis due to blood incompatibility of the Rh factor with evidence of kernicterus prior to dismissal from this hospital; no follow-up has been possible.

Brief summaries of the cases showing unsatisfactory results (death or kernicterus) are presented below; they demonstrate the importance of prompt recognition and early treatment of hyperbilirubinemia.

M. B. This infant was admitted to the University of Kansas Medical Center at 63 hours of age. The birth weight was 3,310 gm. Jaundice had been first noted at 34 hours. The mother's blood type was known to be Rh-negative, and a titer of anti-Rh substances of 1:8 was reported at 7 months gestation. At admission the hemoglobin was 12.4 gm./100 ml. The Coombs test was 4+, and the total bilirubin was reported from the referring hospital as 7.5 mgm./100 ml. At 80 hours the indirect bilirubin had risen to 34.1 mgm./100 ml., and an exchange transfusion was performed. At 88 hours of age the infant was noted to be lethargic. At 92 hours of age the indirect bilirubin had risen again to 26.1, and a second exchange transfusion was carried out. Generalized convulsions occurred at 94 hours, and the child expired on the 6th day after birth. Autopsy on this infant revealed kernicterus.

R. P. This infant was referred to the University of Kansas Medical Center and was 58 hours old on arrival. The birth weight was 3,450 gm. Jaundice had been noted two hours after birth. The maternal blood type was not reported. The admission hemoglobin was 11.5 gm./100 ml. The Coombs test was 4+ and the indirect bilirubin 28.7 mgm./100 ml. Anti-Rh substances were demonstrated. Exchange transfusion was performed at 61 hours of age. The infant was lethargic at admission, with a weak Moro response. Following transfusion the infant became progressively more lethargic, then comatose, and expired 66 hours after birth. Autopsy showed kernicterus and gross bilirubin crystals in the renal pelves.

I. H. This infant was admitted to the University of Kansas Medical Center from another hospital at four days of age. The birth weight was 3,590 gm. Jaundice was first noted "shortly after birth." On admission the infant was extremely icteric, lethargic, and with a poor to absent Moro response. The hemoglobin was 11.0 gm./100 ml. The Coombs test was 4+. The maternal blood type was A, Rh-negative; the blood type of the infant was A, Rh-positive. Immune anti-Rh antibodies were demonstrated. Indirect bilirubin at admission was 19.4 mgm./100 ml. The infant was exchange transfused three hours after admission but expired during exchange. Autopsy showed generalized tissue staining and kernicterus.

L. Mc. This infant was born at the University of Kansas Medical Center. The birth weight was 2,825 gm. The mother was known to be Rh-negative; an anti-Rh titer of 1:16 was noted at the fifth month of gestation. The infant was pale at birth with a cord blood hemoglobin concentration of 5.0 gm./100 ml. and a concentration of indirect bilirubin of 2.0 mgm./-100 ml. The Coombs test was 4+. The infant's blood was exchanged at six hours of age and the patient apparently did well except for rapid respirations. Eighteen hours after birth the infant developed spontaneous pneumothorax, which was relieved by constant suction of the pleural space, but the infant showed increasing respiratory difficulty and expired 52 hours after birth. Autopsy on this infant showed pulmonary hyaline membrane disease with emphysema. There was no evidence of kernicterus.

E. A. This infant had a birth weight of 1,088 grams. With the exception of episodes of apnea of 30- to 90-second duration, the neonatal course was uneventful until the third day when jaundice was noted. The indirect bilirubin at this time was 16.4 mgm./100 ml. Because of the small size of the infant, transfusion was withheld. On the seventh day the indirect bilirubin was 26.5 mgm./100 ml. and it was decided to proceed with exchange transfusion in spite of possible technical difficulty. The weight at this time was 980 grams. Exchange transfusion was performed without difficulty, utilizing the femoral vein. When this child was last observed (at 11 months of age), he was unable to sit without support and showed considerable incoordination of the arms; speech, however, was beginning to develop. It is difficult to know whether or not the present difficulties of this infant are related

to the extreme prematurity, the periods of apnea, or the high concentration of indirect bilirubin.

D. R. This infant was admitted to this hospital at five days of age. The birth weight was 2,870 gm. Jaundice had been noted three hours after birth. On admission the infant was lethargic and the Moro reflex was of poor quality. The admission hemoglobin was 9.5 gm./100 ml. The Coombs test was 4+, and Rh incompatibility was demonstrated. The indirect bilirubin was 16.8 mgm./100 ml.; the total bilirubin was 45.6 mgm./100 ml. Exchange transfusion was performed immediately and was repeated 12 hours after admission. The infant continued to be lethargic but showed gradual improvement in feeding and was dismissed at 15 days after birth. We have been unable to contact this patient for follow-up examination, but the clinical impression prior to discharge was that this child had kernicterus with brain damage.

The age at which clinical jaundice was first noted on the 34 infants born at the University of Kansas Medical Center is shown in Table 3. Jaundice was noted in all of 10 infants with Rh hemolytic disease during the first 24 hours and in 9 of them during the first 6 hours after birth. Jaundice was first recognized in 7 infants with ABO incompatibility at various times during the first 24 hours after birth and in 5 others it was not seen until the second day or even later. None of the patients with idiopathic hyperbilirubinemia were considered to be jaundiced prior to 12 hours after birth, and in most jaundice first appeared after 48 hours.

Table 4 shows the age of the patient at the time exchange transfusion was begun. Infants showing kernicterus and those who expired are indicated by special symbols. Nine of the 19 infants with erythroblastosis due to incompatibility of the Rh group were referred to this hospital. Five of the patients referred here were received 48 to 72 hours after birth, four were received later than 72 hours after birth. Comparison of infants born at this hospital with those referred here reveals 2 of 34 infants born here had unsatisfactory results while 4 of 9 patients referred had unsatisfactory results. Earlier referral for exchange transfusion might have prevented the unsatisfactory results in this group of patients. There were no referred patients in the ABO incompatibility or idiopathic hyperbilirubinemia groups.

Table 3

AGE (Hours)	Rh	ABO	Idiopathic Hyperbilirubinemia
1 - 6	○○○○○○○○	○	
6 - 12		○○	○
12 - 24	○	○○○○	○○○
24 - 48		○○○	
Over 48		○○	○○○○○○○○

Table 3. Age (hours after birth) at which clinical jaundice first reported. Infants born at University of Kansas Medical Center only.

Table 4

AGE (Hours)	Rh	ABO	Idiopathic Hyperbilirubinemia
0 - 6	●○○○○		
6 - 12	○○○	○	
12 - 24	○○	○○○	○
24 - 48		○	
Over 48	●●●◐○○○○○	○○○○○○○	◐○○○○○○○○○

○ = Good Result ◐ = Kernicterus ● = Expired

Table 4. Age (hours after birth) of infants at the time exchange transfusion was started. The term "kernicterus" refers to infants surviving with brain damage.

Table 5 indicates the level of indirect bilirubin in each patient immediately before exchange transfusion. No permanent sequelae occurred in infants exchanged before the concentration of indirect bilirubin in the serum reached 15.0 mgm./100 ml. The infants in the Rh group showing unfavorable results in the 15 to 20.0 mgm./100 ml. range are infants D. R. and I. H. (see case reports).

The incidence of prematurity in idiopathic hyperbilirubinemia is greater than in the other types of hyperbilirubinemia reported here. Four of 12 infants with idiopathic hyperbilirubinemia had birth weights less than 2,500 grams. In the Rh incompatibility group, 2 of 19 infants were premature while in the ABO group one of the 12 infants was premature.

Of the 46 transfusions reported here, 33 (70 per cent) were performed via saphenous or femoral vein cutdown and 14 (30 per cent) via the umbilical vein. The high number of femoral cutdowns reflects the number of exchanges done later than 48 hours after birth as a result of (1) the late referral of nine of the patients to this hospital and (2) the relatively large number of ABO incompatibility hemolytic disease cases and of cases with idiopathic hyperbilirubinemia, both of which conditions are frequently not apparent early in the neonatal course and require transfusion after the umbilical cord is unfit for cannulation. Following femoral or saphenous cannulation there has

Table 5

Indirect Bilirubin (mgm./100ml.)	Rh	A B O	Idiopathic Hyperbilirubinemia
0 - 5.0	●○○		
5.1 - 10.0	○○○○○○○	○○	
10.1 - 15.0	○		○
15.1 - 20.0	●○◐	○○○○	○○○○○
20.1 - 25.0	○○	○○○○○	○○○○
25.1 - 30.0	●	○	◐○○
Over 30.0	●○		

○ = Good Result ◐ = Kernicterus ● = Expired

Table 5. Serum indirect bilirubin concentration immediately prior to exchange transfusion. The term "kernicterus" refers to infants surviving with brain damage.

frequently been cyanosis and edema of the operated extremity, but this has not persisted for more than 48 hours in any case. There have been no permanent undesirable sequelae as a result of cannulation of the femoral or saphenous vein in the patients reported here.

Discussion

The physician who is responsible for the care of newborn infants should be constantly alert for the appearance of icterus during this period. Premature infants with respiratory difficulty must be particularly carefully observed. Once jaundice is clinically evident it must be promptly assessed quantitatively by laboratory determination of the serum indirect bilirubin. Clinical estimation of the severity of jaundice is totally unreliable. When the indirect bilirubin approaches the danger level, prompt exchange transfusion must be carried out or the immediate transfer to a transfusion-equipped facility must be arranged.

No one set of rigid criteria for exchange transfusion is applicable to all cases of hyperbilirubinemia. We have formulated a group of arbitrary criteria which we have found to be a useful guide in determining the need for exchange transfusion. These are based primarily on the work of Hsia and co-workers,[4] of Crosse and others,[5] and Zuelzer and Mudgett.[6] The presence of hydrops fetalis is an indication for immediate emergency exchange transfusion; otherwise, the decision is based on hyperbilirubinemia according to the following rules:

1. A serum indirect bilirubin level rising at a rate exceeding 0.5 mgm./100 ml./hour in the first 24 hours of life *or* exceeding 10 mgm./100 ml. in the first 24 hours.

2. A rising indirect bilirubin approaching 20 mgm./100 ml. at *any* time in the neonatal period. A lower minimum value (16-18 mgm./100 ml.) is employed in premature infants.

3. Inability of the laboratory to demonstrate blood group incompatibility does not alter the decision to perform an exchange transfusion if the indirect bilirubin reaches levels meeting either of the above criteria.

The first of these criteria, dealing with the rate of rise of bilirubin in the first 24 hours of life, is primarily applicable to infants with erythroblastosis due to the Rh factor. These infants show an early and rapid elevation of the bilirubin concentration, while infants with ABO incompatibility and idopathic hyperbilirubinemia demonstrate a later and less rapid rise of bilirubin concentration. The second rule is applicable to all cases of hyperbilirubinemia in the newborn period. Bilirubin determinations may be necessary as often as every six hours until the rate of rise of bilirubin concentration is established, or as infrequently as every 24 hours in some patients, especially those with idiopathic hyperbilirubinemia. It is important to recognize that any such rules are arbitrary and subject to revision to fit each individual patient. Continued observations in hyperbilirubinemia may lead to revision of the criteria stated here.

During the two-year period covered by this study we have exchanged 12 infants who had hyperbilirubinemia without demonstrable blood incompatibility or sepsis to account for the elevated bilirubin. These infants showed jaundice after 48 hours and achieved bilirubin levels comparable to those reached in cases of hemolytic disease. One of these infants shows evidence of kernicterus at 11 months of age.

Brown and Zuelzer[13] have recently reported 46 cases of hyperbilirubinemia without demonstrable incompatibility, and these infants all showed bilirubin levels above 12 mgm./100 ml. None of the term infants in their group showed sequelae, but three infants in the premature group expired with kernicterus having been demonstrated in two of these at autopsy.

It is apparent that this condition, often referred to as "physiologic" jaundice, is not an innocuous condition and deserves corrective therapy (exchange transfusion) to prevent kernicterus. The term "physiologic" jaundice should be dropped from the medical literature as it implies a harmless condition and gives a false sense of security to the practitioner.

Our studies of infants with hyperbilirubinemia have been greatly simplified by the adoption of "microbilirubin" determinations. These permit the determination of bilirubin concentration on specimens obtained by heel puncture and do away with the technically difficult and traumatic venipunctures previously necessary. "Microbilirubins" are done by the method of Powell[15] as modified by Brown and co-workers.[14] These determinations have proved comparable in ac-

curacy to those done by methods utilizing venous blood.

We have not hesitated to perform exchange transfusions when the umbilical vein route is no longer available. Cannulations of the saphenous and femoral veins have proved to be both technically satisfactory and safe in our experience. It may be anticipated that the majority of exchange transfusions for idiopathic hyperbilirubinemia will of necessity be done by femoral cutdown because this condition manifests itself at a time when the umbilical vein is no longer operable.

Summary

1. The purpose of exchange transfusion is to prevent levels of serum indirect bilirubin from exceeding 20 mgm./100 ml.

2. High levels of indirect bilirubin were found in the blood of 43 infants in a period of two years at the University of Kansas Medical Center; 19 infants had Rh-incompatibilities, 12 had ABO incompatibilities, and 12 belonged to the idiopathic group.

3. Determination of the concentration of indirect bilirubin in the blood is essential to the care and management of neonatal jaundice. Bilirubin determinations utilizing capillary blood simplify the problem.

4. The technique of exchange transfusion utilized at the University of Kansas Medical Center is described.

5. It is important that exchange transfusion be performed before irreversible damage to the central nervous system has occurred.

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Philosophy is a product of the humanity of each philosopher, and each philosopher is a man of flesh and bone like himself. And let him do what he will, he philosophizes not with the reason only, but with the will, with the feelings, with the flesh and with the bones, with the whole soul and the whole body. It is the man that philosophizes.

—Miguel de Unamuno

Polycythemia Vera

Study of Coagulation Abnormalities, Both Thrombotic and Hemorrhagic

SLOAN J. WILSON, M.D.; HELEN E. HEATH, A.B., *and* WILLIAM E. LARSEN, M.D., *Kansas City*

An increased incidence of hemorrhage and thrombosis is a paradox in polycythemia vera. The known facts do not give a complete explanation. In the active phase of the disease there is an increase in the red cell mass, hematocrit, platelets, and viscosity, these elements producing a thrombotic tendency. However, the prothrombin and fibrinogen are at least relatively decreased per unit volume of whole blood and contribute to a hemorrhagic tendency.

Stefanini and Dameshek[1] observed a fibrinogenemia of varying severity, the blood clot being unusually small. The small fibrin clot cannot hold the red cells of the elevated hematocrit. The small clot and the falling out of red cells can be mistaken for fibrinolysis. Lawrence[2] also described this peculiar clot retraction with red cell release.

The present study was undertaken to answer the following questions: (1) Is the fibrinogen actually decreased? (2) Is there an increase in fibrinolysis, and is the red cell fall-out phenomenon related to fibrinolysis? (3) Is the phenomenon of red cell fall-out corrected by therapeutic control of the polycythemic state? Previous observations have been published in abstract form by Wilson and Heath.[3]

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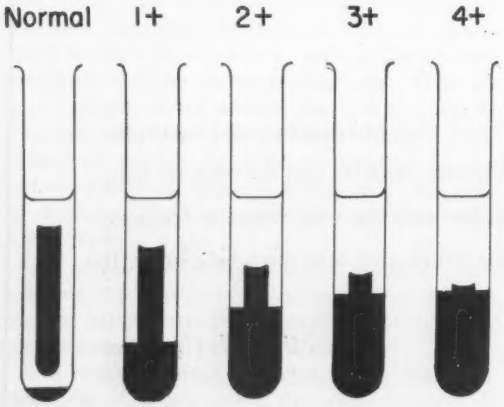

Figure 1. Demonstration of red cell fall-out phenomenon.

Methods

Fibrinolysis was quantitatively determined. Whole blood was collected in sterile siliconed glassware without an anticoagulant, cooled immediately in ice water, and centrifuged at 5° C. The clear plasma was then removed and separated into 1.0 ml. aliquots and incubated at 37° C. Thrombin was added to produce complete and immediate clotting. The fibrin was removed at 0, 24, and 48 hours and thoroughly washed in running water. The amount of fibrin (also equivalent to fibrinogen) was determined quantitatively by the spectrophotometric adaption[3] of the method of Greenberg and Mirolubova.[4]

The study reported here was undertaken to answer three specific questions pertaining to frequent hemorrhagic tendencies in patients with polycythemia vera. The method of study is described, observations are presented and discussed, and conclusions are drawn.

The clot retraction and red cell fall-out phenomenon was observed by placing 5 ml. of whole blood in a test tube and incubating for 24 hours at 37° C.

TABLE 1

DATA, 37 NORMAL SUBJECTS

Average fibrinogen level, mgm. %	310
Range of fibrinogen values, mgm. %	230-402
Fibrinolysis, mgm. % fibrin lysed in 48 hours	
15 subjects (40.5%)	0
16 subjects (42.0%)	2-30
5 subjects (13.5%)	31-60
1 subject (4 %)	75
Red cell fall-out phenomenon	
9 subjects (24.5%)	0 to ±
18 subjects (48.5%)	1 +
10 subjects (27.0%)	2 +

TABLE 2

DATA, 23 TREATED PATIENTS WITH POLYCYTHEMIA VERA

Average fibrinogen level, mgm. %	332
Range of fibrinogen values, mgm. %	229-438
Fibrinolysis, mgm. % fibrin lysed in 48 hours	
10 subjects (43.5%)	0
9 subjects (39.0%)	3-30
2 subjects (8.5%)	31-60
1 subject (4.5%)	108
1 subject (4.5%)	193
Red cell fall-out phenomenon	
4 subjects (17 %)	0-±
3 subjects (13.5%)	1 +
7 subjects (30 %)	2 +
5 subjects (22 %)	3 +
4 subjects (17.5%)	4 +

The degree of separation and red cell fall-out was graded on the basis of 1 to 4+ (see Figure 1). The clot retraction, red cell fall-out phenomenon, and fibrinolysis were done simultaneously.

Observations

For the purpose of comparison, observations were made on 37 normal adult male and female subjects. The results of these observations are tabulated in Table 1.

Studies were completed on 23 patients with polycythemia vera who were in good clinical control. Therapy usually consisted of a combination of radioactive phosphorus (P^{32}) and phlebotomy. One patient was controlled by phlebotomy alone. The results of these studies are tabulated in Table 2.

A third table gives the results of observations on 7 patients with polycythemia who had never been treated.

A comparison of the data in the tables is most interesting. It is to be emphasized that hematocrit values in the treated patients were essentially within normal limits. There is no difference in the fibrinogen values of normal subjects and treated patients with polycythemia vera. The fibrinolysis is also essentially the same with the exception of two treated patients. There is a tremendous difference in the red cell fall-out phenomenon. In 39.5 per cent of the treated patients, the fall-out phenomenon is still abnormal (3 to 4+).

The untreated patients had excellent levels of fibrinogen. It must be remembered that the fibrinogen is measured in mgm. per cent of plasma, not whole blood; therefore, with high hematocrit values there is a relative decrease in fibrinogen. All but two had a 4+ red cell fall-out phenomenon. When under therapeutic control with P^{32} and phlebotomy, one patient exhibited a 4+ phenomenon. Fibrinolysis was not increased with the exception of one patient (F. B.) in whom there was complete lysis. In this instance the fall-out phenomenon could not be evaluated as all fibrin was absent.

Discussion

It is indeed a paradoxical situation when hemorrhage and thrombosis occur in the same patient with polycythemia vera.[1, 2, 6, 7, 8, 9] The incidence of hemorrhage from various areas such as cerebral and gastric vessels is high. The relative decrease in prothrombin and fibrinogen and the increased blood volume and capillary distention contribute to the hemorrhagic tendency.

Previous studies and the present observations in

TABLE 3

DATA, UNTREATED POLYCYTHEMIA VERA

Patient		*Fibrinogen mgm. %*	*Lysis fibrin, mgm. %*	*Red cell fall-out*
S. N.	before treatment	230	0	4 +
	after P^{32} and phlebotomy	260	30	4 +
J. P.	before treatment	282	32	2 +
	after P^{32} and phlebotomy	270	10	4 +
L. K.		300	0	4 +
F. B.		367	367	lysis
W. B.		376	12	4 +
C. B.		220	30	4 +
W. W.		348	19	2 +

untreated patients reveal the inability of the fibrin clot to hold the large quantity of red cells, and the fall-out phenomenon is marked. However, we feel that the previous explanations are totally inadequate inasmuch as 39.5 per cent of our treated patients still retained a defective fibrin clot with persistence of the red cell fall-out phenomenon. Our interpretation is that the fibrin (and possibly the fibrinogen) is at fault. There could even be an abnormality in the molecular structure.

Increased fibrinolysis is rare, having been noted in only two treated patients and in one untreated patient. Thus fibrinolysis does not explain the fall-out phenomenon. No patient in our series had a decrease in fibrinogen such as has been reported in an occasional patient with polycythemia vera.

Conclusions

Certain coagulation factors were observed in patients with polycythemia in an attempt to explain further the frequent hemorrhagic tendencies.

Fibrinogen levels, fibrinolysis, and the red cell fall-out phenomenon (separation of red cells and clot) were observed in 37 normal subjects and 30 patients with polycythemia vera (23 treated and 7 untreated).

The fibrinogen levels and the degree of fibrinolysis were essentially the same in normal subjects and patients with polycythemia vera. Increased fibrinolysis was observed in only two treated and one untreated patient.

The inability of the fibrin clot to hold red cells (resulting in a fall-out phenomenon) was striking in untreated polycythemia vera. Therapy did not correct this defect, 39.5 per cent of treated patients retaining this phenomenon. This suggests a defect in the fibrin itself and possibly in its precursor fibrinogen. The extremely poor fibrin strands probably contribute to the hemorrhagic tendency.

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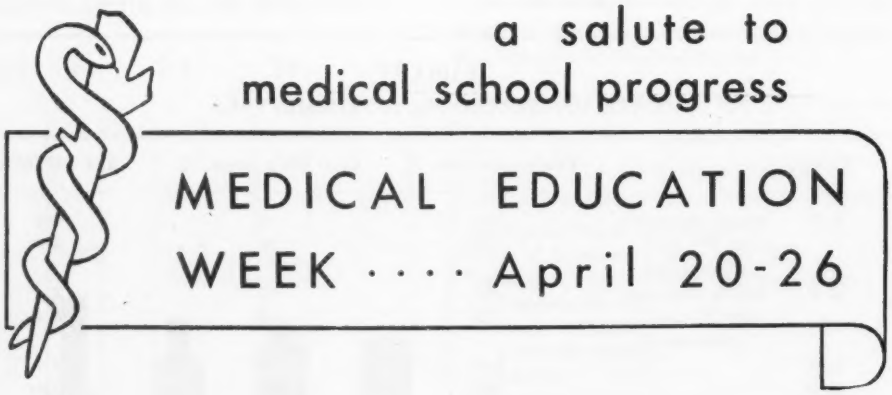

Arteriovenous Fistula

Report of Case Developing after Operation for Ruptured Intervertebral Disc and Successfully Repaired by Homograft

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Arterial-venous fistulas have been reported following trauma or surgical procedures. Elkin[1] comments on the frequency by relating a personal series of 650 operations on aneurysm and arteriovenous fistulas with a 1 per cent incidence following previous surgery. Linton and White,[2] and Glass and Ilgenfritz[3] have reported arteriovenous fistulas secondary to operation for intervertebral disc.

These cases were treated by quadruple ligation with a preliminary sympathectomy. The great vessels lying on the anterior surface of the lumbar vertebrae are in a vulnerable location. Plunging through the anterior annulus in removing a ruptured intervertebral disc may injure the underlying arteries and veins. This unfortunate accident is accompanied by severe blood loss.

The effects of a developing arteriovenous fistula on the cardiovascular system have been aptly summarized by Pratt[4] and Holman.[5]

1. A fall in arterial pressure with a gradual recovery, the systolic higher and the diastolic lower, reflecting a greater pulse pressure.
2. Increased venous pressure.
3. Increased pulse rate.
4. An increased cardiac output, depending on the site and size of the fistula.
5. Temporary decrease in the size of the heart followed by an increase and hypertrophy of the heart.
6. Increased total blood volume.
7. Development of collateral circulation around the fistula and, if the above process continues, uncorrected cardiac failure.

Case Report

A 35-year-old white female was admitted to the University of Kansas Medical Center on May 15, 1954. Six months prior to admission she was in an auto accident. She complained of non-radiating pain low in the back associated with numbness and weakness of the left leg. Myelograms were made, and an operation for a ruptured intervertebral disc was performed elsewhere. The patient states she "lost a lot of blood" during the operation. Three months prior to admission to the University of Kansas Medical Center she complained of a swollen abdomen, nausea, emesis, and dyspnea on exertion. After three weeks of hospitalization, she returned to her home with generalized weakness and ankle edema even though she was digitalized.

A case of arteriovenous fistula secondary to operation for ruptured intervertebral disc was successfully repaired with a homograft. This procedure cured the patient from the symptoms following a traumatic arteriovenous fistula.

Physical examination revealed a chronically ill dyspneic female with pitting edema of the ankles and the chest wall. Blood pressure was 115/65. Pulse rate was 90. A low grade fever appeared every other day, reaching 100.5°F. Jugular vein pulsated synchronously with the carotid pulse. Basilar bilateral rales were heard. A bruit was auscultated over the lower abdominal aorta and transmitted to both iliac arteries. An apical systolic Grade II and Grade III murmur was present with a diastolic gallop over the apical and mitral areas. The heart was enlarged with a point of maximal impulse in the anterior axillary line. The liver and spleen were palpable and tender. Pistol shot sounds were heard over the left femoral artery.

Laboratory studies revealed a normal complete blood count, nonprotein nitrogen, creatinine, blood sugar, and serum electrolytes. Wassermann was negative. Blood cultures were negative. Prothrombin time was 60 per cent of normal. Urinalysis showed occasional pus cells. A. aerogenes was cultured from the urine. Blood volume studies showed an increased plasma volume and a total cell mass.

Electrocardiogram showed a rate of 100 with digitalis effect. P waves were peaked in leads II, III, and AVF. Chest x-rays confirmed cardiac enlargement and pulmonary congestion (Figure 1).

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Figure 1. Heart size of patient on admission showing cardiac enlargement and pulmonary congestion.

A lumbar aortogram done with 70 per cent Urokon localized a fistula between the left iliac artery and vein.

The patient was prepared for surgery with salt restriction, digitalis, vitamin K, oxide, Gantrisin for urinary infection, and daily procaine penicillin.

Operation was done on May 21, 1954, under continuous spinal anesthesia. An arteriovenous fistula was found on the left, 2 cm. from the iliac bifurcation. The fistula measured 1 cm. in diameter. The proximal and distal iliac arteries were mobilized and cross-clamped. When the proximal iliac clamp was applied, a transient Branham's sign with a bradycardia of 50 developed. The proximal and distal vein was compressed, and the fistula was excised. The iliac vein was sutured in a linear fashion. The posterior wall of the iliac artery was missing with surrounding scarring and secondary arteriosclerotic changes. The iliac artery was resected for a distance of 5 cm., and the defect was replaced with a frozen one-week-old homograft (Figure 2). Anastomoses were accomplished with 00000 black silk suture. Occluding clamps were removed restoring arterial and venous blood flow. Blood pressure during surgery ranged from 160-140/80.

Figure 2. Frozen homograft spanning surgical defect common iliac artery.

The postoperative course was dramatic in the patient's improvement. She was ambulatory on the third postoperative day. Blood volume and venous pressures returned to normal. Normal left femoral and distal pulses were present. Postoperative digitalis and antibiotics were continued. The electrocardiogram showed a marked decrease in rate from 100 to 50. T waves were inverted in leads IV, V_2, and V_3, indicating rotation of T vector to the left. The patient was discharged on May 29, 1954. Followup examination three years after surgical repair revealed a return to normal in heart size, disappearance of systolic heart murmur, and no signs of cardiac failure (Figure 3). The patient has good femoral and distal pulses with no claudication on exercise or walking.

Figure 3. Reduction of heart size following closure of A-V fistula and grafting of iliac artery.

Comment

The surgical correction of arteriovenous fistulas has usually been quadruple proximal and distal ligation of the arteries and the veins. Important considerations in such a procedure are the age of the patient, anatomical location of the fistula, and the potential collateral circulation capable of being developed after sudden ligation. Acute ligation of the common iliac artery carries a reported incidence of 6 per cent[6] to 50 per cent[7] gangrene to the distal extremity. Patients treated surgical-

ly for arteriovenous fistulas in the lower extremities by ligation, with or without sympathectomy, may have varying degrees of exertional claudication.

The recent availability and familiarity of arterial grafting procedures offer a better solution to the problem of arteriovenous fistulas—especially, as in this case, when the fistula is located in the common iliac artery and vein. Anatomical repair by graft replacement will insure a direct arterial flow and eliminate the probability of claudication.

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Deafness

Examination Techniques for Evaluating Malingering and Psychogenic Disabilities

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During the last 10 to 15 years there has been an increasing awareness on the part of physicians of the need for more precise techniques to evaluate claims of hearing disability resulting from accidents in industry. The otologist, or specialist in pathology of the ear, is well aware that the standard otological examination is frequently not adequate to determine whether or not the patient has suffered injury to the auditory mechanism. The elaborate equipment necessary to show that the injury to hearing either has or has not been sustained is usually to be found at a university hospital.

However, it happens occasionally that the otologist in the field is not utilizing to full potential the equipment which may be at his disposal. The purpose of this paper, therefore, is to review current techniques which are employed in university hospitals to expose feigned hearing loss with special emphasis on tests which may be performed on a two channelled speech pure tone audiometer.

The individual who feigns a hearing loss as a result of a fall, blow on the head, or other such accident which occurred during working hours stands to receive compensation by virtue of the disability. In general, malingerers professing hearing loss fall into two general classifications, i.e., those with feigned bilateral auditory impairment and those with feigned unilateral hearing impairment. The two classifications may be further sub-divided into degrees of malingered hearing loss. Thus, the individual may feign a bilateral total or a bilateral partial hearing loss, and, similarly, a unilateral total or a unilateral partial hearing loss.

Various tests for detecting functional deafness have been outlined. Specific attention was given to tests which could be performed in an otologist's office.

Still another factor with which one is confronted in the examination of medico-legal cases is the frequent presence of organic hearing loss, the magnitude of which has been exaggerated by the individual. Whether or not the underlying organic impairment is associated with the accident is difficult at times to establish. The case history covering the patient's age, his previous occupations, recreational habits, activity in military service, if any, previous illnesses, and family history of deafness usually contribute enlightening information in lieu of final test results.

Tests to Detect Bilateral Malingering

Prior to the development of the delayed feedback test, there was no satisfactory method for quantitatively exposing the individual who feigned total bi-

lateral deafness. The Lombard test afforded a method for securing indications of malingering from relatively naive malingerers, but it too frequently leaves the issue in doubt in the case of sophisticated individuals. The test is based on the reflex tendency of a person to raise the sound intensity of the voice in the presence of noise as compared to the level employed in quiet. As an example, one might have the patient recite a short rhyme or read out loud a passage from a book, noting, during the execution of the task, the loudness of the patient's voice. Next, apply Barany noise makers to the ears of the patient (one on each ear) and have him continue to recite or read out loud. Observe whether his voice increases in loudness during the recitation in noise as compared to the recitation in quiet. Quite frequently naive malingerers will literally shout under the noise condition.

Since development of the two channelled pure tone audiometer, equipped with a controlled masking source, the Lombard test can be done with much better precision. This may be illustrated by the following procedure. Place the air conduction receivers on the patient and have him either recite or read out loud from a pre-selected passage. Note the loudness level of his voice. Gradually introduce the masking simultaneously into his ears through the earphones. Note the level at which the patient first begins to raise his voice. Continue to increase the level of the noise until the maximum loudness is reached. Observe whether the patient continues to raise his voice level as increments in noise are added. The naive malingerer will often be shouting at the top of his voice when the sound is at its maximum intensity. Gradually withdraw noise from the two earphones, noting during the procedure the decreasing loudness of the patient's voice. Check the level at which the voice returns to the pre-noise exposure loudness.

If an average level of noise at which the reflex to raise as well as lower the voice has previously been determined on the equipment, by averaging the values at which the phenomenon takes place on about ten subjects with normal hearing, then a rough quantitative measure is afforded for determining the degree of malingering. This test may be done in the otologist's office or single test room. It does not require a two-room set-up.

Unfortunately, many malingerers soon become aware of the nature of the test and learn to modify the loudness of the voice under noise exposure. Differences then between the noise and the quiet conditions of reading or reciting may be either missing entirely or so minor as to leave a diagnosis one way or the other in doubt.

Another simple method for obtaining information relative to whether or not a patient is feigning total bilateral deafness is to inadvertently (at least make it appear so to the patient) put either maximum noise or a pure tone (we use 4000 cycles per second because of its irritating effects) in both ears and then leave the room on some pretense. From a pre-determined vantage point observe the behavior of the patient. Frequently the patient will furtively watch the door for re-entrance of the examiner and either remove the earphones completely from the head to reduce the din or else hold them as far away from the ears as the head band allows. If one purposely catches the patient in the act, he offers a weak excuse often pertaining to the pressure of the earphones. This particular technique for exposing the patient who malingers bilateral total deafness has been used by otologists since time immemorial. At best, when successful, it indicates to the otologist that the patient is prevaricating with reference to total deafness. No information is provided relative to the extent a patient may be exaggerating a moderate organic loss.

One of the best methods for detecting bilateral total hearing less is the Delayed Feedback Test. The phenomenon of haltering and stuttering which takes place when one's own voice is fed back through earphones to the ears under certain conditions of delay during the course of reading or reciting has been rather extensively studied. Many commercial audiometers such as the Beltone 15A are equipped so that a double headed tape recorder may be attached which records the speech of the individual under test and plays the speech back through earphones (worn by the subject) at about a .16 second delay. Recent experimental work with delayed feedback by Hanley and Tiffany[16] has indicated that changes in speech occur as low as 20 decibels relative to the speech reception threshold. In other words, the effects of delayed speech upon the individual under test are discernible when the level of the speech is only slightly above the threshold of speech. When the delayed speech is 40 decibels or more above threshold, the effects upon the individual under test are even more pronounced. It is possible, therefore, with the delayed feedback test not only to get quantitative evidence of malingering but also to specify within limits the degree of loss if there is one.

For example, a patient purporting to have bilateral total absence of hearing who manifests severe disturbance in speech at 40 decibels relative to the normal threshold of speech, would be exposed as possessing relatively either normal or near normal hearing (within 20 decibels of normal) in at least one ear. Subsequent examination with monaural delayed speech, testing each ear separately with masking on the opposite ear, affords a method for checking the ears individually, thus determining whether or not a unilateral impairment exists. This test will be described under unilateral tests.

Another technique, utilized extensively in the diagnosis of feigned and psychogenic hearing loss, is the psychogalvanic skin test or PGSR. Unfortunately, in the light of increasing research, PGSR audiometry does not appear to be the panacea its more optimistic proponents envisaged. Goldstein and his associates[11, 12, 13] in a series of studies have reported that approximately half of their subjects with normal hearing, whether children or adults, were difficult to condition with the galvanic skin test. Further, Goldstein has reported on the difficulty of conditioning often encountered in adults suspected of functional hearing loss. These investigations do not imply that the PGSR test is of no value in the diagnosis of hearing loss. Rather, they stress the importance of the skin-resistance technique as a valuable adjunct in differential diagnosis, and point up emphatically that interpretation of results must be made with extreme caution and in the light of research findings to date.

Johnson, Work, and McCoy[18] advocate the use of PGSR audiometry in testing for functional overlay, but they feel that the results must be considered in conjunction with other methods. The equipment required for the test is usually to be found only in university and hospital clinics. However, the equipment is commercially available and built for use with many of the clinical audiometers which are now on the market. Although a two-room set-up is not required for this test, it seems unlikely that the average otologist could spare the time required either to master the technique or to administer the test. Again, a team of two persons is necessary when small children are involved.

The repeat audiogram test, on the other hand, provides a method which is of practical use to the otologist in the field. The technique is of no avail in cases of feigned bilateral total hearing loss. Information of a general nature may be obtained when the patient feigns bilateral partial deafness. Gundrum[15] recently compared the test-retest audiograms of 50 subjects with normal hearing who were requested to simulate bilateral partial deafness with 50 patients who possessed bilateral partial hearing loss. In the former group only two subjects were able to simulate reasonably well the thresholds of their first test. The latter or second group (true organics) showed only minor variations between the two tests. It would appear therefore, from the results of this study, that the repeat audiogram technique affords a method for determining the presence of malingering in cases of bilateral partial feigned deafness.

In addition to the evidence relative to malingering and functional deafness which is provided by the patient's inability to repeat the threshold reasonably well, there are certain other manifestations pertaining to characteristics of the audiogram and type of response which bear discussion. Further, the relationship between the obtained speech reception threshold and the average pure tone threshold for frequencies 500-1,000-2,000 cycles per second frequently contribute valuable information with reference to psychogenic deafness.

Some years ago, Doerfler,[5] in lieu of the development of the D-S test,[7] pointed out that the characteristic pure tone air conduction curves in about 80 per cent of patients with functional deafness were saucer-shaped and tended to range between a 50-decibel and a 90-decibel level. The term "saucer-shaped" refers to the similarity between the audiograms of functional loss patients, whether psychogenic or the result of malingering (hence either on an unconscious or a conscious basis) and the equal loudness contours or phone lines. Individuals with normal hearing who simulate deafness will usually manifest a pure tone air conduction audiogram which is remarkably similar to the 60-decibel equal loudness contour. In other words, they either consciously or unconsciously balance the loudness of test frequencies at a level which corresponds roughly to the comfort loudness listening level of the normal ear. The occurrence, then, of the saucer-shaped audiogram at a 60-decibel level in conjunction with other manifestations subsequently to be discussed, is strongly suggestive of normal hearing. When there is functional overlay, or an exaggeration of an organic flat hearing loss, the obtained audiogram might be expected to lie in the region of the 90-decibel equal loudness contour, depending, of course, upon the degree of organic involvement.

Johnson, Work, and McCoy[18] recently illustrated the emergence of the saucer-shaped audiogram in patients with simulated deafness whose true hearing thresholds were normal except for a dip at 4,000 and 8,000 cycles. The explanation for the saucer audiogram or equal loudness contour lay in the recruitment at the aforementioned two frequencies. As the curves, after repeat audiograms, approach the actual threshold, the saucer characteristic disappears, as would be expected.

The appearance then of a saucer-shaped audiogram, particularly when there are other evidences of functional deafness such as delayed and slow responses, restlessness, a general evasive attitude on the part of the patient in consistency in test-retest responses, poor bone conduction responses inconsistent on retest, etc., must be treated with extreme suspicion.

As previously noted, the relationship between the speech reception threshold and the pure tone air conduction average loss for 500-1,000-2,000 cycles per second is often an important indicator of psychogenic deafness. Differences between the speech reception threshold and the average pure tone air conduction loss, as indicated above for flat audiograms, are re-

garded as insignificant when the magnitude is of the order of 6 decibels or less. Therefore, a speech reception hearing loss of 45 decibels with a pure tone average loss by air conduction of 51 decibels would be within the limits of accepted normal variation. If, however, the discrepancy between the two aforementioned thresholds lies between 7 and 13 decibels, with the speech threshold being the better, there is strong suspicion of psychogenic deafness. Differences which exceed 13 decibels (speech being the better) are practically first order evidence of psychogenic deafness and, hence, psychiatric referral.

The D-S test[7] affords another method for uncovering functional hearing disorders. The test, however, necessitates a two-room set-up. Doerfler and Stewart observed that patients with functional hearing loss manifested an exaggerated disturbance in speech reception in the presence of noise. To illustrate, a person with either normal hearing or an organic hearing loss will not succumb to the masking effects of noise upon speech until the level of the noise is about 20 decibels greater than the level at which the speech is being presented. For example, a person with normal hearing or an organic hearing loss (provided discrimination is not extremely poor in the latter case) will repeat spondees with 100 per cent efficiency when they are administered in quiet at a level 5 decibels or more above the speech reception threshold. If noise is introduced and gradually increased, ability to repeat the spondees will not be completely obliterated until the level of the noise is 20 decibels greater than the level at which the speech is being presented.

However, with the psychogenically deaf patient, such is not the case. Doerfler and Stewart observed that in cases of functional deafness, speech reception was disturbed and in many instances was completely obliterated when the level of the noise was 10 to 15 decibels weaker than even the admitted speech reception threshold. Thus, a disturbance in speech reception by noise which is actually too weak to produce a masking effect provides evidence of functional deafness.

When the test is administered the level of the noise is gradually increased until it has exceeded the level of speech presentation by 20 decibels or more. (In other words, to a point at which masking of speech is 100 per cent effective.) The level at which the speech was given is then lowered by 20 or 25 decibels. Gradually the noise is attenuated until the lower level of speech again emerges through the noise. Malingerers as well as psychogenics, their loudness points of reference upset by the noise, will begin to respond again, but this time at a level 20 to 25 decibels below their former admitted reception level for speech.

In the foregoing paragraphs, many of the techniques currently employed to detect bilateral total and partial functional hearing loss were discussed. The succeeding pages will be concerned with tests of unilateral total and partial hearing loss.

Unilateral Tests

Evidence of unilateral feigned deafness is at once apparent when, in the course of the pure tone air conduction test, the audiogram of one ear is either normal or relatively so with complete absence of response on the opposite ear. The reason, of course, is that a shadow curve will appear in the case of organic unilateral total deafness. It is a well established fact that the shadow curve, or cross hearing in the aforementioned type of hearing loss, takes place at about the 50-decibel level. Further evidence of malingering is obtained when the patient vociferously claims inability to hear the bone conduction tone applied to the supposedly deafened ear. As is well known, the shadow bone curve in unilateral organic deafness occurs on an average at a 10-decibel level. As a general rule, using air conduction, failure of a response curve to appear at between 50 and 60 decibels relative to the obtained threshold in the admittedly good ear as well as failure of the bone response curve of the supposedly deafened ear to appear at a level 10 to 15 decibels greater than the recorded bone curve of the better ear, strongly suggests feigned deafness.

Regardless of whether or not there are evidences for suspecting simulated hearing loss, every patient with either unilateral total or partial impairment is administered a battery of tests designed to expose the malingerer.

The unilateral Lombard test can be performed in a few minutes and, in the case of naive malingerers, often helps to confirm initial indications of simulated deafness. The patient, after donning the headphones, is requested to either read out loud a passage from a book, recite a poem, or in some instances count to 100. The level of the voice is noted. Suddenly, noise of about 80 decibels is introduced into the supposedly deafened ear. A marked increase in the voice level will be in evidence if the hearing loss is simulated. Subsequently, the noise is switched back and forth between the ears. If the increased level of the voice is maintained regardless of the ear under stimulation, malingered deafness is indicated. If, on the other hand, the voice is apparently raised only when the noise is channelled to the admittedly good ear, organic deafness of the claimed deafened ear is indicated. Unfortunately, the malingerer of average ability soon learns to modify his voice in the presence of unilateral noise, thus vitiating the effectiveness of the test.

The Stenger test first described in 1907 is within

certain limits one of the best techniques for exposing simulated unilateral deafness. However, Gibbons and Winchester[10] have recently emphasized several drawbacks of the test which may lead to spurious results. When technical failure of the equipment is concerned, as manifested by a difference in the time element of tonal presentation to the ears (in our experience associated with audiometers of slow tonal rise), by frequency differences producing a beat, as well as by electrical cross-over from one channel to the other, we are in full agreement with Gibbons and Winchester. However, in the absence of technical failure, which in our experience has been achieved in the Beltone 15A audiometer, an additional objection, i.e., namely, unfamiliarity with the principles of the test as a prerequisite for its success, is groundless. As a matter of fact, it has been our practice to thoroughly explain the principles of the Stenger to our graduate students in audiology and otology and then have them attempt to literally "beat the test." We have found the test unbeatable.

The Stenger test is associated with the principle that when tones of identical frequency but differing as to intensity are presented simultaneously to the two ears, only the tone dominant in intensity will be heard, the weaker tone being masked out. Our administration of the Stenger test, using tones, is always executed under the guise of rechecking the admittedly good ear. If, for example, the threshold of the good ear lies at 5 decibels at 1,000 cycles per second, this threshold is rechecked. During the recheck however, the tone is presented at threshold plus 5 decibels to assure 100 per cent response. The attenuators are set then so that there will be a simultaneous presentation to the supposedly deaf ear and good ear at a 40-decibel and 10-decibel level respectively.

Absence of a response is definite evidence that the tone is being heard in the supposedly deafened ear. The attenuator controlling the stimulus to this ear is lowered in 5-decibel steps until a response finally occurs. If the patient does not respond until the intensity of the tone is equal to the intensity of the tone in the admittedly good ear, there is definite evidence that the ears are approximately equal in sensitivity. By repeating the same procedure at other frequencies, an audiogram may be obtained on the ear which will approximate the true threshold. If, on the other hand, the patient responds at the initial settings of the attenuators (hearing loss deals) i.e., 40 decibels and 5 decibels, then there is the possibility of a true organic loss of the ear in question. To check this possibility, the tone is removed completely from the admittedly good ear. On re-introduction of the signal, therefore, only the supposedly deafened ear is being stimulated. If the patient responds, he is definitely hearing the tone in the ear. Conversely, if there is no response, an organic loss of the ear is indicated. The procedure is repeated a number of times to verify the results.

It sometimes happens that a patient simulating a severe or total unilateral loss will tend to exaggerate the threshold even in the admittedly good ear. An initial audiogram of the ear may therefore show a 10- to 15-decibel depression. Usually, the examiner is aware of the situation by virtue of the nature of the patient's response pattern (threshold variability, hesitancy of response, etc.). With the procedure as described in the preceding paragraphs, it is often possible to determine the relatively true threshold. For example, the patient whose threshold response on the good ear is at 15 decibels and who has failed to respond with simultaneous presentation of the tones to the ears at 40 decibels and 20 decibels will often respond to a final stimuli-presentation of, for example, ODB to the supposedly deaf ear and 5 decibels to the good ear. Confusion as a result of the first stimuli-presentation is more than likely to bring a true response when the tone is definitely in the good ear. Thus, the Stenger test which is usually thought of as a technique for exposing unilateral malingerers may also be employed to detect bilateral partial malingering.

It will be noted, in the examples we have cited, that the initial stimulus to the supposedly deafened ear has been at a 40-decibel level over the normal threshold. The reason for the level is to insure that the tone to the affected ear is below the level of cross hearing. To be more explicit, there is always danger of cross-hearing when the intensity of the tone exceeds 50 decibels in the event that there is actually some organic loss with superimposed simulated deafness. Such a condition which is not infrequent would obviously confuse the test results. Unilateral organic hearing losses of 40 decibels or so are at times exaggerated to the 70- or 80-decibel level. In cases of this type, the use of the Stenger test becomes hazardous. The unilateral delayed feedback test, to be discussed, should be the technique of choice.

The principles of the Stenger test which have been emphasized in conjunction with the pure tone test are also applicable to the modified Stenger test using spondee words. Therefore, a detailed description of this procedure will not be given. Suffice to say that in our clinic, the modified Stenger is an indispensable part of the battery of tests which are used to uncover functional deafness. It is particularly well-suited for the examination of children. Although the modified Stenger test can be done much more effectively in a two-room set-up employing monitored level voice, because of its flexibility, nevertheless we have recently been experimenting with the tests using records (W-1

and W-2). Results have been gratifying. At present research is under way to experimentally determine the usefulness and limits of the test in this form. If this research should demonstrate the validity of technique, another method will be available to the practicing otologist, since a two-room set-up would not be required for its administration.

Within the last year Winchester and Gibbons[10, 27] have experimentally demonstrated that the unilateral delayed side test (delayed feedback test) with noise (saw tooth) in the opposite ear is an effective method for determining functional deafness. Essentially, the test consists of a comparison between the time required to read a selection of prose of at least 500 syllables (the selection is homogeneous as to difficulty and at a fifth grade level) with 60 decibels of delayed speech on the good ear and 80 decibels of noise on the apparently deaf ear and the time required to read the same passage with the stimuli conditions reversed (60 decibels of delayed speech to supposed deaf ear and noise on the good ear).

Gibbons and Winchester[10, 27] advocate the administration of the tests as follows:

Step 1. Obtain organic thresholds, including speech reception threshold for the better ear.

Step 2. Screen the subject to establish his ability to read at or above the level of the material used.

Step 3. Give instructions regarding the conduct of the test. Care should be taken that the subject understands what is expected of him, especially that he is not to stop or hesitate while reading.

Step 4. Present a sensation level of 60 decibels of delayed sidetone to the better ear while at the same time presenting a masking noise level of 80 decibels over normal threshold to the apparently poorer ear.

Step 5. Signal the subject to begin reading the test material which should consist of at least 500 syllables of easily read expository prose. The elapsed reading time should be measured, perferably through the use of a stop watch.

Step 6. Reverse the above conditions so that the same level of delayed sidetone is presented to the apparently poorer ear, while masking noise at 80 decibels over normal threshold is presented to the better ear.

Step 7. Signal the subject to begin the same test passage used previously; elapsed reading time should be measured as before.

In the interpretation of results the authors have stated that a difference of more than 10 seconds between the readings is strongly indicative of organic deafness. (The condition of 60 decibels of delayed speech on the poorer ear and noise 80 decibels on the good ear would give a shorter reading time.) When the difference between the readings is smaller than 10 seconds, a functional element is suspected. Aside from reading rate, marked changes in speech production contribute to the diagnosis. We have found the unilateral delayed speech test an invaluable adjunct to our battery of tests for functional hearing loss. It is particularly useful in exploring unilateral organic losses of about 40 decibels upon which a functional element has been superimposed. As previously pointed out, Stenger tests are not reliable in such cases. Provided the practicing otologist has the equipment, the delayed feedback tests can be done in a one-room set-up.

Another test to detect unilateral feigned hearing loss is the swing test. One method of administering the test is to tell the patient a story. On pre-determined words of the story during the narration to the good ear, the speech is swung to the supposedly deafened ear (good ear silent). The patient is then questioned on the content of the passage. Success indicates that he has heard in the supposedly poor ear, since the questions are designed so that they cannot be answered unless the key words to the apparently poor ear have been heard. We have not found this test to be as effective as many of the others which were discussed.

Other methods for exposing functional deafness such as the use of narcosynthesis have been described in the literature. We have attempted, however, to review the techniques which are generally utilized in hearing clinics with emphasis on those which can be done without a two-room audiological set-up.

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The Retarded Child

Fifty Years Ago a Custodial Problem—Now One in Education and Rehabilitation

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Fifty years ago the integration of the exceptional child into school and community was a less complex affair than it is today. There was only one kind of retarded child widely recognized then; he was the child who called attention to himself because of the extreme severity of his retardation. Many other children with less severe handicaps did not take well to book learning. These children eased out of school situations and found successful adjustment as handymen and domestics, so greatly in demand in a non-mechanized society.

Even the severely retarded child was not recognized then as either a medical or an educational problem. He was entirely a custodial problem. Families provided custody for their own severely afflicted children when they could, and there were large homes with many rooms to facilitate this home care. When home care was not possible, the state providentially took over, adding another cot to an institution euphemistically referred to as a training school. These institutions had very little to offer, certainly neither training nor school, except for the fact that they were in an inaccessible part of the state, thus effectively removing the child as a social problem.

This picture has changed. Unhappily we are still left with some of the same physical plants or asylums, which strive to be training schools, and we are still left with some of the old attitudes toward retarded children—the attitudes as archaic as the buildings. The change was effected by a numer of events.

First, the social and economic structure became more complex, more urban-centered. The less seriously retarded, high-grade mental defectives found it more difficult to escape clinical and social recognition.

Second, the development of better psychometric devices and their widespread application to troops during World War I revealed a mass of previously

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The physician who is called upon to diagnose or treat a retarded child has an obligation to the patient's parents as well as to the patient. An attempt is made to point out areas in which the art of medicine can enhance the practice of medicine in such situations.

unrecognized illiterate, uneducated, and intellectually subnormal persons to a surprised society.

And, third, universal compulsory education began to approach reality. It became less easy for the child who didn't take kindly to learning to ease out of school into a job. Most surprising of all, the compulsory part of universal education underwent a switch, largely through the efforts of parents' groups organized since World War II. Society had previously readily accepted the concept that school was compulsory for all children; now society proposed to compel schools to accept all comers—to educate or train all children to the limit of their potential, no matter how meager that might be. There are indications that this philosophy will prevail.

Before examining the physician's responsibilities in the management of exceptional children, some facts about these children should be reviewed.

First, most retarded children can benefit enormously from education and can lead fruitful and independent lives. About 75 per cent of all retarded children are in the high-grade category with intelligence quotients ranging between 50 and 75. School systems have designated these the "educable group." Doubts as to how "educable," in the sense of successful social adjustment, this group is, can be dispelled by reference to a study done in Connecticut a number of years ago.[1] Records on 250 moron children (we would now call them educable retarded) were secured from the 1937 census files of "social inadequates" and were matched in every way with a group of non-moron children. The two groups were similar in family size, income, religion, social, and economic level; in fact the two groups appeared identical except for their "educability." All persons in the study were born between 1915 and 1925. In 1944 the typical moron and the typical non-moron appeared as follows:

1. They each had a spouse and one child.
2. They were employed and self-supporting.
3. They were skilled or semi-skilled workers.
4. They earned $35 to $55 per week.
5. They had held the same number of jobs.
6. Their housing arrangements were the same.
7. They were saving money, were paying for some kind of insurance, owned radios (I'm sure it would be TV sets today), and they had telephones in their homes.
8. They had never been arrested and had no court records.
9. They habitually read a newspaper and a magazine.
10. Their participation in group activities and military service was the same.

Twenty years later, there was no major difference in the social adjustment of the child who was in a special class for retarded children and the average child in an average classroom. The abundant economy of the period when this study was made may have contributed to the easy adjustment of the handicapped persons.

How does the retarded child fare during the school years? Not so well. A recent study on the incidence of retarded children in two counties in the East showed the following.[2] In children under 4 years of age, the incidence of retardation appeared to be 0.1 per cent. With increasing age, the incidence rose to a high of 4.4 per cent in the 10- to 14-year age group and then fell off to 0.8 per cent in the older age groups. The highest incidence in the school age groups indicates that diminished verbal ability and abstract reasoning of these children call attention to their handicap in an academic setting, but not so often in pre-school and post-school life situations. There is danger here that frustrations and failures during school years, will, unless expertly handled, produce long lasting emotional maladjustments in persons with a fairly respectable potential for productive and successful social adjustment. Here is an important consideration. Exceptional children make unexceptional adults, for compensations can be made for all but severe forms of retardation. A child with the double handicap of retardation and emotional disturbance cannot make the compensations necessary for successful socialization.

The origins of emotional disorders of retarded children can be found usually in one of two places: (1) in the failure of parents to adjust to the handicapped child, and here physicians can be of enormous preventive and therapeutic aid; and (2) the frustrations of an inappropriate school placement.

Occasionally, one hears the view expressed that retarded children "deserve the opportunity" to enroll in regular classrooms. This usually means that they must painfully demonstrate their inadequacies to the world and to themselves before they can be enrolled for special education. This invites disaster in the form of chronic failures and resistance to any further attempts at education. If a child is known to be retarded, he needs specialized education which individualizes his program to the maximum degree. He may be greatly harmed by repeated failures in school; he will not be particularly benefited by running several times through a kindergarten program in which he is not able to participate.

There is a handicap in addition to the retardation which hinders the successful integration of these children into school and community. Retarded children are denied our best professional skills. A diagnosis of retardation automatically excludes children from therapy in many child guidance clinics. An intelligence quotient of 150 rates a psychologist's report three

times as detailed as that for a child with a rating one third as high. Many physicians discharge their obligations to retarded patients in a hasty and embarrassed fashion. And social case-work for retarded children is too often limited to assisting parents in filling out forms for institutionalization. Educators preceded other professional people in recognizing their obligations to retarded children, but in most professions concern with retardation has only recently begun to move a little upward on the "totem pole of professional respectability."

We have referred to "educable" retarded children and have accepted the definition of this group in terms of intelligence quotients. Such testing reveals only a small part of the child; personality, motivation, health, and a great many other factors are important and sometimes decisive in estimating a child's achievements. At least we talk this way about most children. When considering retarded children, the intelligence quotient acquires a validity we would never grant it under other circumstances—so much so that it is accepted by most school systems as the sole criterion for class placement of retarded children.

In point of fact, the reliability of psychometric testing is greatest near the average range. Deviation away from this range is associated with a high degree of error. The best opinion of psychologists today is that conventional psychometric tests have not been adequately standardized in the range below 70. Our own experience has been that in this range repeat testing will reveal deviations as great as 25 points.

We credit psychometric testing with a validity it does not deserve, using it, I think, to disguise our inability to arrive at sound judgments on any other basis. Speech therapists have said that the only way they can determine whether or not a retarded child will benefit from special training is to try it, usually for as long as six to nine months. If the same philosophy were applied to other training endeavors, we might be surprised by favorable results.

Psychologists are not the only professional people beginning to examine their own inadequacies. Children having educational problems commonly are referred to their physicians. Deviant behavior and aberrations in development are regarded as medical problems, and rightfully so. Retardation is mimicked by deafness, psychiatric disorders, aphasias, and poor physical health. Great importance in management and interpretation attaches to correct diagnosis.

Physicians may be discouraged by the small number of cases for whom they can make a positive contribution. There are more than 125 different medical conditions which are associated with mental retardation. Yet, we usually are unable to say with confidence which of these conditions, if any, pertain to a specific retarded child. The number of retarded children who can be benefited by medical treatment is exceedingly small. This should not be interpreted to mean that retardation is a problem outside the province of the physician. In spite of our limitations, the physician is the key figure in diagnosis, treatment, and counseling.

Perhaps the physician's chief role is as a counselor to parents. There are few things so shattering to family stability as the knowledge that a child is retarded. The resistance of parents to accept their child's handicap, their fear of social stigma, or their refusal to participate in management programs can disturb their own mental health as well as their child's. No less disturbing is the parent who believes that if a normal child can learn something with an ounce of effort, a retarded child can learn the same thing with a pound of effort. They attempt to compensate for their child's handicap by long and tedious hours of drilling. They succeed only in diminishing the child's true potential by creating emotional disturbances and antipathies toward subsequent more controlled efforts at education.

Some of the most important ways of influencing a parent's attitude toward mental retardation and specifically toward his own handicapped child are the physician's techniques of history taking, physical examination, and investigative work-up, and the interpretation of these findings to parents. That these procedures may have an adverse effect on parental attitudes may be attested to by the great numbers of parents who "shop" from one physician to another, or one "healer" to another, not receiving the satisfaction and assurances they need, and becoming increasingly hostile and resistant to medical opinion.

Several principles may be followed in the study of any child suspected of being mentally retarded. Ample time should be provided. A frequent complaint made by disappointed parents is, "How does he know our problem? He was with us only a few minutes." The few minutes may be sufficient to diagnose mongolism and to render a reasonably accurate prognosis, but they are not sufficient to inspire confidence nor to learn the problems of a family.

Whenever possible, both parents should be included in the study sessions. Too often one parent is left to defend a medical opinion to a hostile spouse with unanswered questions of his own.

Attempts to "sell" an idea, or to convince parents of the truth of a diagnosis or a prognosis, or of the advisability of a certain disposition, usually are not successful and serve only to promote parental resistance. Careful review of the developmental history and comparison of accomplishments of the patient with his chronological peers will indicate the handicaps to the parents as well as to the doctor.

Average parents are aware that their child is retarded, though they seldom mention this in presenting their complaints. Once parents are helped to express their beliefs or fears, especially if the study has substantiated them, the physician should be unhesitant in confirming them. Equivocation, unnecessary delay, and false reassurances are disservices equal to too much hasty and ill-considered direct advice. Frequently parents receive the news that their child is retarded with a sense of relief; they have known this for many years but have been unable to get professional confirmation.

Many parents, in general accepting their child's handicap, cling to the notion of his superiority in some narrow achievements, usually mechanical abilities, and usually destructive ones, such as dismantling clocks. This is frequently interpreted by parents as scientific curiosity. Parents of older retarded children are likely to give histories of normal early motor development. These may be true. With the passage of time, many parents need to convince themselves that they produced a normal child and gave him a normal start in life; that the world has somehow changed him to a retarded child.

Considerable time should be spent in discussing probable etiologies. Parents want an explanation for their child's retardation. A definite etiologic diagnosis can seldom be given. It is important in these instances to dwell at some length on etiologies which are felt *not* to pertain. Some parents are comforted by learning that no known deficiencies of diet or care during pregnancy were at fault, and that no amount of skilled medical care has succeeded in preventing most kinds of retardation. Parents need to be educated a little about the frequency and kinds of retardation; and they need to know a little about our ignorance of the causes. In planning for the care of a retarded child, parents find it helpful not only to accept the child and his deficiencies but to accept the fact that medical science may have nothing to offer that will substantially alter the developmental pattern of the child.

This brings us to a consideration of what *can* be done about it. Parents have not only the right but also the responsibility for deciding what is to be done for or with their child. This right is respected by the physician. However, the physician has the responsibility to help the parents think through their problems. He does this by considering with the parents all possible resources and plans of action and the effects these plans may have on all members of the family.

The problem of institutional care should certainly be discussed, even when this seems like a remote prospect to parents and physician. It is likely that someone, perhaps another physician, will at some time urge parents to put their child in a training school. Parents should have explored this course of action sufficiently that such urging will not threaten their family security. So few people can afford the fees of private schools that institutionalization usually means placement in a state school. This may be equated in the minds of parents with "confinement" or "putting the child away." The whole concept may be so repugnant that parents may cling to a child who might actually better be cared for in a training school. Other parents may react with less vehemence but with equal determination in feeling that they are duty-bound to care for their own child—to live with their shame. Parents can be spared these reactions if the physician presents to them the idea that state schools are resources, which fortunately are available and which should be taken advantage of, when that plan seems advisable, just as public schools are resources for most normal children.

The "training" aspect of state schools may hold a false attraction for some parents. The training may be excellent, but I doubt that it is ever as helpful as a loving family environment and a special community program. In actuality, most children are in training schools for some reason in addition to their retardation. That reason is commonly a disrupted family.

Intact families making plans for a retarded child should weigh the advantages of state school placement, along with other resources, not only insofar as the needs of the retarded child are concerned but also considering the total needs of every family member. The final decision should be influenced as little as possible by guilt at "putting the child away," or by fears that normal children will be adversely affected by association with their retarded sibling.

These are fears related to institutionalization which are frequently encountered. There are many others which should be ventilated before the family can make any final decisions. The physician's role should be to support them in their decision, not only at the time of the initial work-up but on a continuing basis. The child's parents benefit from sympathetic understanding and gradually develop a more complete acceptance of the reality of their situation.

Together with the continuing support which parents receive from their physician, they get tremendous support from each other. Many communities now have organizations for parents devoted to the education of retarded children or pertaining to them. Here they learn the extent of the problem; the fact that the misfortune affects families irrespective of economic, social, and intellectual attainment, that their problems are being faced and perhaps solved by others. They learn to talk about their own problems, to let the skeleton out of the closet. Wherever these

parents' groups are in operation, parents who are suitably prepared may profitably be referred to them.

There are a number of popular misconceptions about retardation which are important to correct whether one is working with individual parents or groups of them. One of these misconceptions has to do with understanding mental age. Occasionally a parent has been told that his child is functioning at 50 per cent of his expected performance, and that he will always do so. This parent may find false comfort in the thought that his child at 30 will have the mental capacity of a 15-year-old, and that won't be so bad.

More frequently parents are told that their child will ultimately achieve the mental age of, say, a six-year-old. They interpret this as meaning that their child's skills and fund of knowledge will never be greater than a normal six-year-old child's, and that his accomplishments will cease after that time. An adult who has had the mental age of a six-year-old for a great many years is not the same as a first-grade child. Average adults have the mental age of 14-year-olds. This does not mean that new knowledge and new skills have not been acquired throughout life. Neither do retarded children reach a saturation point. They continue to learn, but at their own reduced rates.

Parents are apt to tack the word "special" in front of training, and think that educators have developed techniques, different from those used with normal children, that will miraculously correct retardation. This unfortunately is not so. Parents are apt to feel that their young child is retarded only because his muscle control is poor, or that a somewhat older child appears retarded only because he doesn't speak well; that if through *special* training the child could be taught the mechanics of talking, he would say smart things. These are notions that cannot be easily turned aside, and there is no formula for answering them except patient understanding.

Finally, many parents and some professional people have misconceptions about the training goals for retarded children. Parents need help in understanding that the usual school curriculum is not their child's dish, even at a decelerated pace. Retarded children, through diligent effort, may acquire a reading vocabulary of say 300 to 400 words, after which they can be handed a comic book. Is it not more important for these children to read "stop" and "go," "men" and "women," to count change, to drive a nail straight, and to meet people graciously?

We talk as though all retarded children are educable, or trainable, and in some measure they are. The child whose developmental pace keeps him at the infantile level is no less a community medical problem than his less severely damaged neighbor. We cannot assume that all low-grade defectives will be cared for in institutions. (And even if they were, their parents' needs for support would not be lessened.)

Institutionalization is the least satisfactory and the most expensive means of caring for retarded children. Our state institutions can accommodate about one retarded child in 20; and the fact remains that most parents prefer to care for their handicapped child in the family unit. This is fortunate so long as parents can be helped to apportion their energies, affection, and money wisely. We cannot turn these responsibilities over to social workers, welfare agencies, special classes, or visiting nurses, even where these resources exist. These people can be of tremendous help, but they too will rightfully look to the family physician for guidance.

Dr. Grover Powers has written: "Should not the physician act on the precept that a doctor is a doctor at all times and in all situations where there is suffering, and not find interest and resource solely where cure is probable! Should not he develop skills and technics here as in other areas of medical practice whereby he may help parents in their hour of black despair and offer them requisite time and encouragement to talk over implications and potentialities and possibilities and also give them insight into community resources?"

An enlightened and agitated public is knocking at the doors of our offices, hospitals, and schools demanding that something more be done for handicapped children. Something shall be done. Physicians in cooperation with other professional people must see that the best care is given and that community leaders are helped to establish the best programs. If this responsibility is shirked, we invite quackeries and waste of public and private funds on easy solutions which will work a hardship on physicians, teachers, and, most important of all, on a large and deserving group of children.

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39th and Rainbow
Kansas City 12, Kansas

Acknowledgements

Some of the material in this paper was taken from the following sources:

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Primary Carcinoma of the Vagina

Results of Treatment by Radiation

CHARLES A. HUNTER, JR., M.D., ***Kansas City***

Primary carcinoma of the vagina is a rare malignancy of the female genital tract which has been attended by a high mortality rate. Of all gynecological cancers, malignancies of the vagina rank second only to Fallopian tube carcinomas in the lowest percentage of five-year survivals. Because of the infrequency of this disease, little attention has been given the subject.

This presentation is a review of all proved cases of primary malignancies of the vagina seen at the University of Kansas Medical Center in the past 25 years (1932-1957). A few cases, initially thought to be primary carcinoma of the vagina, were excluded from this series after further study proved them to be primary carcinomas of the cervix with the vagina secondarily involved.

The purpose of this review is to analyze our own material critically and report the results of therapy.

Incidence.—During this 25-year-period, 33 proved cases of primary carcinoma of the vagina have been treated at the University of Kansas Medical Center. Within the same period of time, 1,512 patients with gynecologic cancer were admitted to this institution. The incidence of primary carcinoma is 2.2 per cent of all female genital tract malignancies.

Age.—Ages of patients in this series range from 32 to 84 years, with the majority in the sixth and seventh decades of life at the time of the original diagnosis (Table I).

Parity.—Table II shows the distribution of parity in these 33 patients. Approximately three-fourths of the patients had delivered children.

Symptoms and Clinical Course.—Symptoms are bleeding or brownish-color discharge as a result of ulceration of the vaginal mucosa. Thirty patients in this group stated that these were the first symptoms. In the remaining three, the initial symptom was finding a "lump in the vagina." Most often, the initial bleeding is insidious. In two patients, however, the initial bleeding was so severe it required them to seek medical attention immediately. Symptoms of pressure or pain in the pelvis are late to appear.

A critical summary of 33 cases of primary carcinoma of the vagina treated at the University of Kansas Medical Center in the past 25 years has been presented. All were treated with radiation which has resulted in an absolute five-year cure rate of 18.5 per cent—a shockingly low cure rate. The major factor responsible for the universally poor results of this disease is discussed. The complete extirpation of all cancer-involved tissues when applicable is recommended as primary treatment for primary carcinoma of the vagina. Radiation therapy should be reserved for those not amenable to these radical surgical procedures. It is hoped by such a program that this disease will yield results comparable to those of other malignant lesions of the female genital tract.

The average duration of symptoms before treatment was 6.3 months. The extremes of this period were one day in two patients and 30 months for another. Reports in the literature showed a similar period of delay. Often these tumors reached considerable size before producing the initial symptom of bleeding.

TABLE I
AGE DISTRIBUTION

Age	*No. of Patients*
30-39	3
40-49	5
50-59	7
60-69	12
70-79	3
80 & over	3

TABLE II
PARITY

Parity	*No. of Patients*
0	8
1	6
2	3
over 2	15
unknown	1

TABLE III
LOCATION OF PRIMARY VAGINAL LESION

Upper Third of Vagina		13
Anterior wall	7	
Posterior wall	6	
Middle Third of Vagina		5
Anterior wall	4	
Posterior wall	1	
Lower Third of Vagina		9
Anterior wall	5	
Posterior wall	4	
Annular		3
Unknown		3

Location of the Tumor.—Although the growth may be located anywhere in the vagina, the majority of authors state that the most common location of the primary vaginal carcinoma is in the upper third of the vagina. In the present series, 13 (39.4 per cent) involved the upper third of the vagina and 9, or 27.4 per cent, involved the lower third. In three cases no definite location of the tumor could be determined due to rather extensive involvement of the vagina. An annular type of growth, which encircled the vagina, was noted in three instances (Table III). The location of the tumor in the vagina has important therapeutic considerations and will be discussed later.

The smallest vaginal tumor described was 1 cm. in diameter, and the largest was depicted as "the size of an orange." In most cases, size was not adequately described.

Lymphatic Drainage.—The lymphatic drainage of the vagina may be divided into three parts: upper, middle, and lower third (Figure 1). The lymphatic drainage of the upper third of the vagina is similar to the cervix: parametrial; obturator; hypogastric; and iliac nodes. The mid-portion of the vagina is drained primarily by the perivesical lymphatics to the external iliac chain. However, Way[1] points out that tumors in this area may drain in either direction. In the lower portion of the vagina, the lymphatic drainage is similar to the vulva and involves the superficial and deep inguinal nodes. The lymphatics surrounding the vagina freely anastomose and much overlapping of the lymphatic drainage is apparent. It has been demonstrated that a primary tumor located in the upper third of the vagina may involve the inguinal lymph nodes without involvement of the lower half of the vagina. A clear knowledge of the lymphatic drainage of the vagina is essential as a basis of treatment.

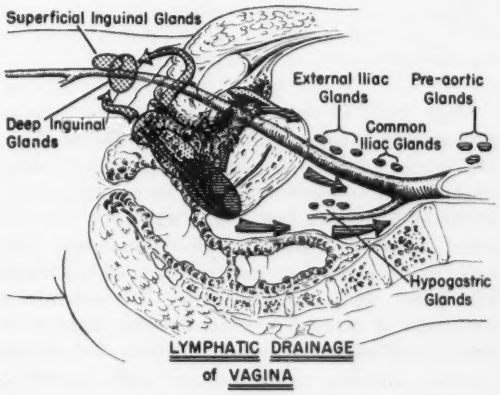

Figure 1. Lymphatic draining of yagina.

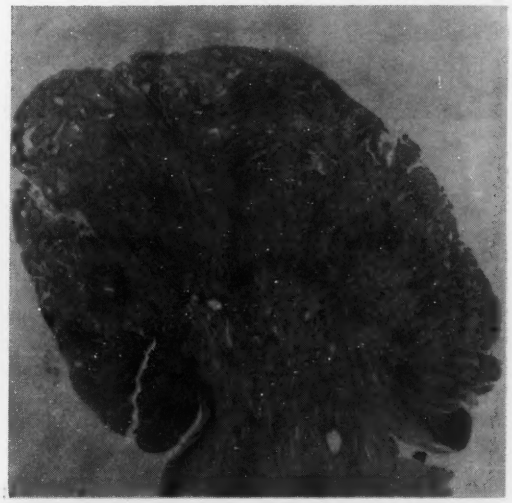

Figure 2. Everting or papillary type of epidermoid carcinoma of the vagina.

Pathologic and Histologic Characteristics.—Gross characteristics of these tumors have been classified by Healy[4] into three groups: everting or papillary carcinomas; infiltrating or inverting carcinomas; and annular or surface tumors. All three types were encountered in this series.

Everting tumors are the most frequent type encountered, according to the literature. Some authors state that this type of tumor outnumbers other forms by 2 to 1. There were 19 cases of everting tumors in this series. These tumors are characterized by a polypoid growth of carcinoma projecting above the surface of the surrounding epithelium. Microscopically, the edges of the tumor are fairly clear cut and have a tendency to show minimal invasion in the immediate underlying tissue (Figure 2).

The infiltrating type of tumors clinically appear as firm, ulcerative lesions with considerable induration into the submucosal tissues. The tendency for lateral

Figure 3. Infiltrating type of epidermoid carcinoma of the vagina.

growth in this lesion has been clearly described by Douglas.[5] As shown in Figure 3, the infiltration of the submucosal tissues with groups and cords of tumor cells is much greater in area than would be presumed by the size of the surface lesion. Accompanying this diffuse tumor infiltration, there is a marked inflammatory reaction which tends to accentuate the limits of this induration. As suggested by Way,[1] Douglas,[5] and Smith,[6] the induration of the pelvic tissues surrounding this malignancy may clinically be much greater than the actual spread of the tumor. The clinical significance of this finding will be discussed later.

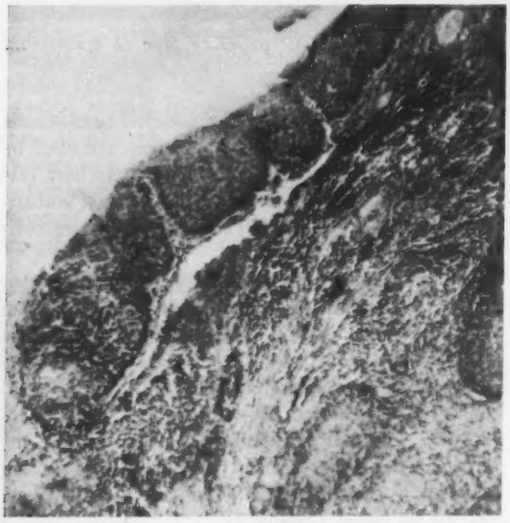

Figure 4. Annular type of epidermoid carcinoma of the vagina. Note the thickening of the vaginal epithelium.

The annular type of tumor growth is manifest clinically by firm induration encircling the vagina and tends to develop ulceration of the mucosa rather late in the disease. Douglas described one case in which he found the malignancy mainly a surface lesion with a large portion represented as intraepithelial in extent. Two cases in this series show marked field changes of the basal cells in the vaginal mucosa (Figure 4 and Figure 5).

All of our 33 cases of primary carcinoma of the vagina are epidermoid carcinomas. Most of these tumors are histologically moderately or highly undifferentiated, only five showing a tendency to keratinization and pearl formations. No attempt was made to grade the tumors into degrees of differentiation.

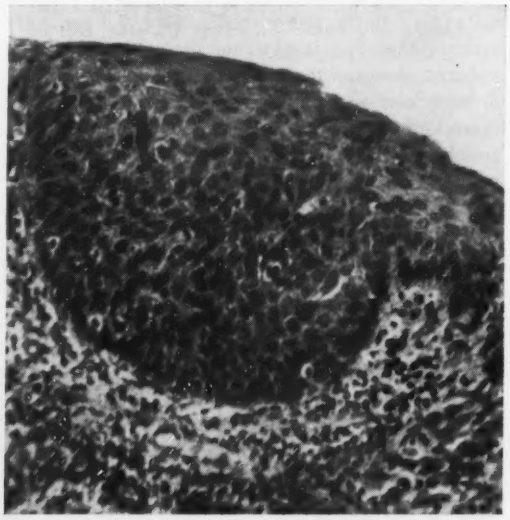

Figure 5. Annular type of epidermoid carcinoma of the vagina. Higher power view of Figure 4. Note the marked field changes of the basal cells.

Stage of the Disease.—Attempts to find a satisfactory method of staging this malignancy have not been successful. Courtial[7] divided his cases into three stages: Stage I—tumor limited to the vagina and not involving more than half of the wall from which it arises; Stage II—tumor extending over more than half of the anterior or posterior vaginal wall with perivaginal soft tissue infiltration, but without involvement of bladder or rectal mucosa; Stage III—tumor infiltrating the rectum, bladder, or urethral mucosa, extending to the vulva or with fixation to pelvic bone.

Recently Douglas[5] has raised serious question as to the adequacy of determining the extent of spread of primary carcinomas of the vagina by clinical palpation. In a small but well documented study he reported failure to find regional node metastasis in any of his operated cases in which nodal involvement was suspected clinically. The "clinically involved nodes" revealed only evidence of inflammation, probably secondary to inflammatory diseases in the vaginal tissues surrounding the tumor. Likewise, he stated that if spread is suspected from thickening of the rectovaginal septum or parametria, the likelihood of inflammatory induration should be kept in mind.

In view of the above discussion, the cases in this series are divided clinically into two groups: (1) tumor localized to the vaginal mucosa or involving the immediate surrounding tissues and not fixed (localized); (2) tumor extended to involve the bladder or rectal mucosa or fixation of tumor to the bony pelvis and palpable inguinal node involvement (advanced).

Twenty of the 33 patients had only clinical involvement of the immediate surrounding tissues of the vagina and would fit into the above described localized stage. By such a staging, it would seem that this would be the favorable group.

In the unfavorable or advanced stage there were 12 patients. This group included those with fixation to the bony pelvis, palpable inguinal node involvement, or evidences of distant metastasis. Two patients had distant metastasis to the bone by x-ray evidence. One had metastasis to the lumbar vertebrae and is still alive 11 years after her initial therapy. The other patient had metastasis to the femur. Other terminal cases had metastasis to the lung, liver, and adrenals proved by autopsy.

Treatment and Results

The primary treatment of carcinoma of the vagina has been by radiation in most of the larger cancer clinics. In this group of patients, 32 had only irradiation therapy. In one patient extensive (but not radical) pelvic surgery was performed, followed by deep x-ray therapy.

Of the 32 patients treated with radiation, six received only external deep x-ray therapy to the pelvis. Three patients were treated by external deep x-ray therapy to the pelvis plus intravaginal x-ray therapy. One patient was treated with intravaginal radium application alone. The amount of radiation given to a patient has varied widely in this series, which may be partially accounted for by the fear of fistula formation because of the proximity of the bladder and rectum.

The five-year survival rate among the 27 eligible cases in this series is 25.9 per cent. If the two patients who are alive but with evidence of cancer are excluded, the absolute five-year "cure" rate would be 18.5 per cent. This is compared to Courtial[7] who reports the highest five-year survival rate of 45.4 per cent in a series of 22 patients with primary vaginal carcinoma. Other reports in the literature vary from 0 per cent[8] to 27.5 per cent.[9]

Complications resulting from therapy have been rather high in this series. The most frequent complication is radiation sickness, which has been classified as marked in 9 patients. The subsequent development of rectovaginal fistulae occurred in four patients, and three developed vesicovaginal fistulae. One patient developed a large perirectal abscess. Five patients were subjected to a colostomy as a palliative procedure.

Discussion

It is apparent from this study that our management of primary carcinoma of the vagina leaves much to be desired. We have been lulled into thinking of this disease as being similar to the more commonly seen problem of carcinoma of the cervix. The pitfalls in this reasoning become apparent when the anatomical location and the lymphatic drainage are considered. As Stanley Way[1] stated, "... when we meet a small carcinoma of the vagina involving the bladder, we tend to think of it as being hopelessly advanced. Now this is not always the case."

The radiation administered to these patients has varied widely, and we did not always deliver a cancericidal dose to the tumor. Even today, there is no commonly accepted method of delivering adequate radiation to these tumors. There are wide differences of opinion among radiologists concerning the problem of total dose, techniques of application, and duration of therapy for treatment of carcinoma of the vagina. Perhaps with newer radiotherapeutic methods these problems may be overcome.

Recently a few of the larger cancer clinics have been employing radical pelvic surgery, in selected cases, as primary treatment of this tumor. If one considers the above comments on the lymphatic drainage and stage of the disease, it becomes obvious that any surgical procedure would, by necessity, have to include at least complete colpectomy, radical hysterectomy, and radical pelvic lymphadenectomy. If either the bladder or rectum is involved, removal of the involved organ will be necessary. Certainly if the primary cancer is located in the lower third of the vagina, due consideration of radical excision of the inguinal glands would be important. These operative procedures are formidable and must be individually planned with the aim of producing a cure. The applicability rate of this type of radical surgery is

limited because of the large number of poor-risk patients. Smith,[6] Way,[1] and Douglas[5] have reported their results of radical surgery for treatment of primary carcinoma of the vagina. Admittedly, the combined series represent a small number of patients, but the results appear encouraging when compared to the low salvage attained by radiation therapy.

Until satisfactory radiotherapeutic methods are developed, the author believes that every woman with this disease should be considered as a possible candidate for radical pelvic surgery as primary treatment. For patients in whom surgery in contraindicated, well administered intravaginal radium and deep x-ray therapy should be given.

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The family of man has been in existence for a long time. Civilization is twelve thousand years old, they tell us, and that there may have been prior civilizations. The Hebrew religion is somewhat short of six thousand years old; and Christianity a little less than two thousand.

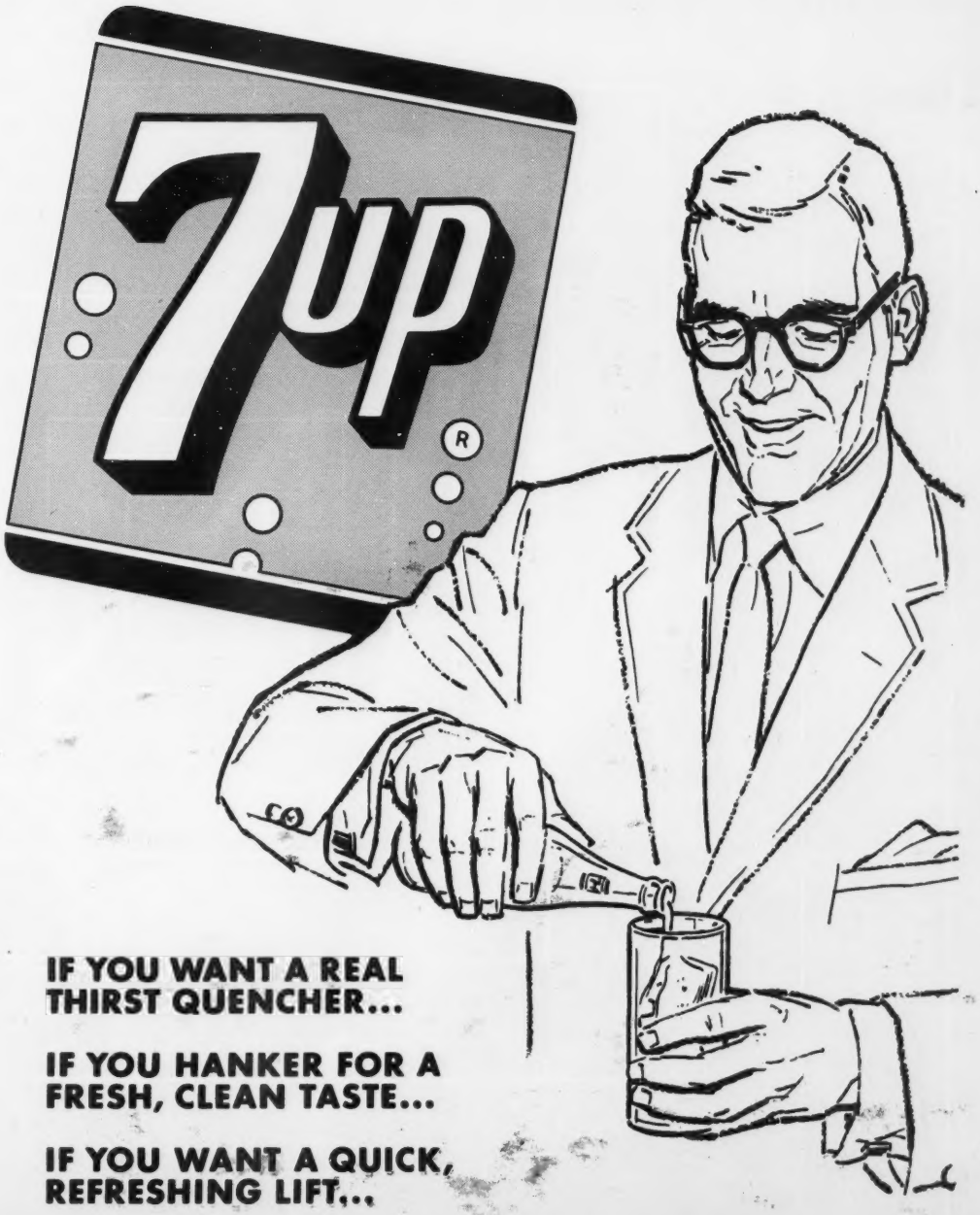
This (February 16-23, 1958) is the twenty-fifth anniversary of Brotherhood Week. Twenty-five years in six, twelve or two thousand are as a split second in eternity.

Man cannot live by bread alone. The things of the spirit are the only imperishables we acquire in this short life journey. Brotherhood is not a matter of belief or teaching . . . far too many of us believe, but do not *know*. There is a difference. Few of us really learn. Brotherhood is a quality of the spirit.

Lip service is not enough. Tolerance . . . that dreadful word . . . of one another's beliefs is not enough. Brotherhood must be as deep rooted in the spirit as a natural involvement, outwardly expressed in service as the worship of God.

How profoundly I wish and pray that when the fiftieth anniversary of Brotherhood Week comes, *there will be no need for it!*

—*Faith Baldwin*



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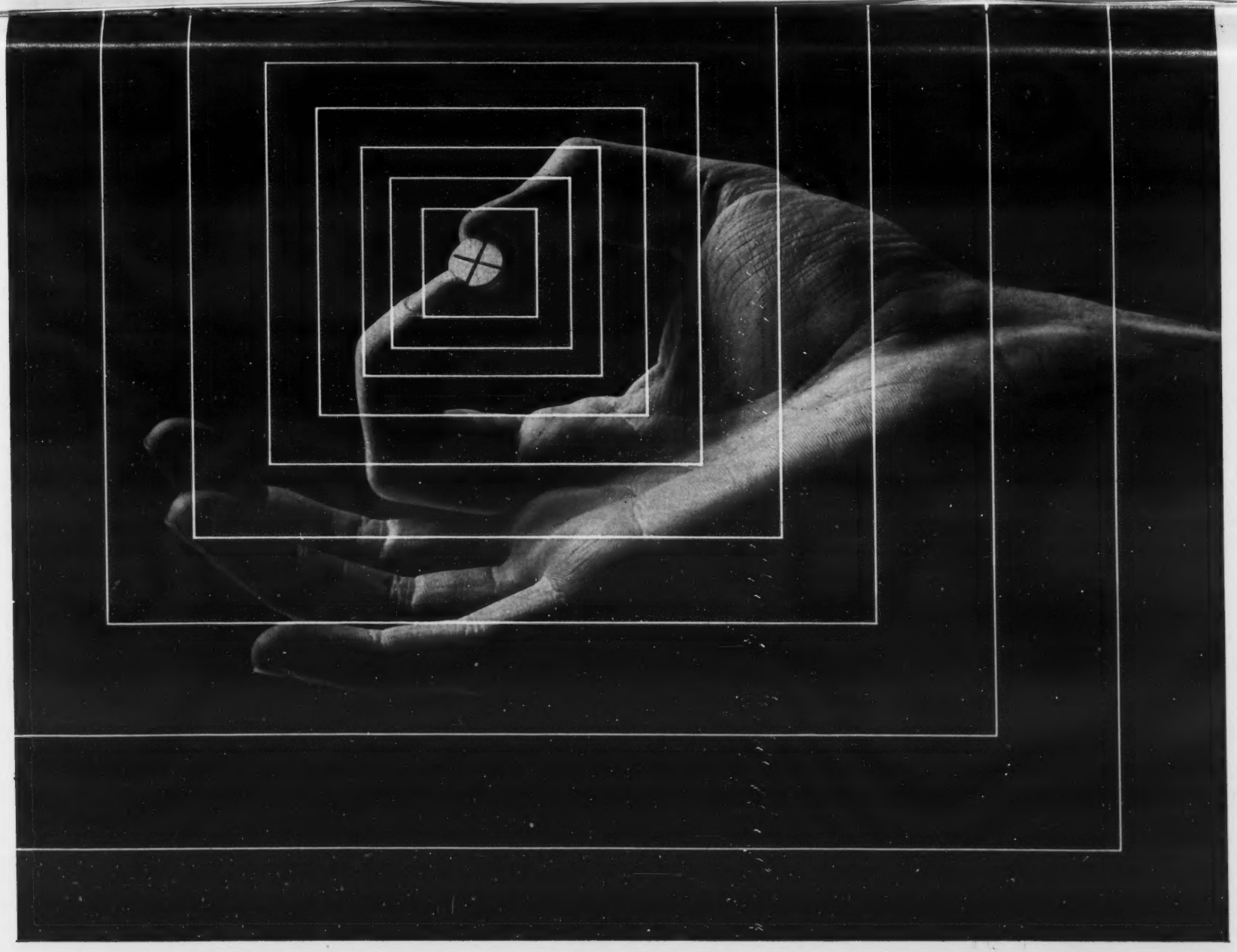

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Asian Influenza Vaccine

How Effective Was It in Prevention of Acute Respiratory Infection at the University of Kansas Medical Center?

ROBERT A. JORDAN, M.D., *and* TOM D. Y. CHIN, M.D., *Kansas City*

During October and November 1957, an epidemic of acute respiratory illness affected nearly 40 per cent of the more than 2,000 students and employees at the University of Kansas Medical Center. At the same time a similar outbreak of respiratory illness spread through the Greater Kansas City area.

Specimens consisting of ten throat washings and seven paired sera were obtained from ten patients seen at the Student Health Service of the Medical Center during the peak of the epidemic. Asian influenza was demonstrated in seven of these patients. The Asian strain of influenza virus was recovered from the throat washings in six patients and four-fold or greater increase in hemagglutination-inhibition (HI) or complement-fixing (CF) antibodies was detected in five patients.

In addition, 14 throat washings obtained from 22 patients living in the Greater Kansas City area also were positive for Asian influenza virus. On the basis of these findings, it appeared that a large proportion of the respiratory illnesses occurring during this period were actually Asian influenza.

Since roughly 50 per cent of the Medical Center students and employees were inoculated with Asian influenza vaccine and the remaining group did not receive the vaccine, it appeared worthwhile to compare the incidences of respiratory illness in these two groups as a means of estimating the effectiveness of the vaccine.

Methods

Although some 200 cases of acute respiratory illness were treated during this period at the Medical Center Health Service, a much greater number of patients consulted private physicians, or administered self-treatment at home. In order to collect sufficient data for study, a questionnaire was prepared for the purpose of obtaining the following information: whether or not the individual had recently (since August 15, 1957) suffered an influenza-like illness; the date of onset, duration, and symptoms experienced in any such illness. A check-list of symptoms was printed on the questionnaire. Each person was also requested to state whether or not he had received the Asian influenza vaccine, and, if so, to give the date of inoculation.

In a study of the effectiveness of vaccination with 200 C.C.A. units of the Asian virus in an epidemic of acute respiratory illnesses occurring among employees and students at Kansas University Medical Center during the fall of 1957, it was learned that 63 per cent were protected. Details of the study are presented.

These questionnaires were sent to 2,000 employees and students during the week following December 10, 1957. All of the various departments and student groups at the Medical Center were included. The student groups included 142 student nurses, most of whom reside in the Nurses' Home which provides for two students per room. The survey also included 309 medical students; however, none of them live in large dormitories or rooming houses. Most of the remaining students and employees at the Medical Center reside in private homes or in apartment houses. No attempt was made to classify the Medical Center population into age groups; however, there were no individuals less than 18 years of age in the study.

Approximately 1,000 employees and students were inoculated with the Asian influenza vaccine at the Student Health Service of the Medical Center during the first 15 days of October 1957. A monovalent vaccine containing 200 C.C.A. units per ml. of the A/Japan/-305/57 strain of influenza virus was administered in a dose of 1.0 ml. subcutaneously. Several persons obtained the vaccine from other sources (approximately 6.0 per cent of the total, according to the questionnaire data).

From the Communicable Disease Center, Bureau of State Services, Public Health Service, U. S. Department of Health, Education, and Welfare; Department of Internal Medicine, University of Kansas Medical Center, Kansas City 3, Kansas.

Dr. Jordan is Assistant Professor of Internal Medicine and Director of Health Service. Dr. Chin is Assistant Chief, Kansas City Field Station, Communicable Disease Center, PHS, Kansas City, Kansas.

Acknowledgment is made of the technical assistance of Clifton R. Gravelle, Kansas City Field Station, and Jean Weston, Hixon Memorial Laboratory, University of Kansas Medical Center.

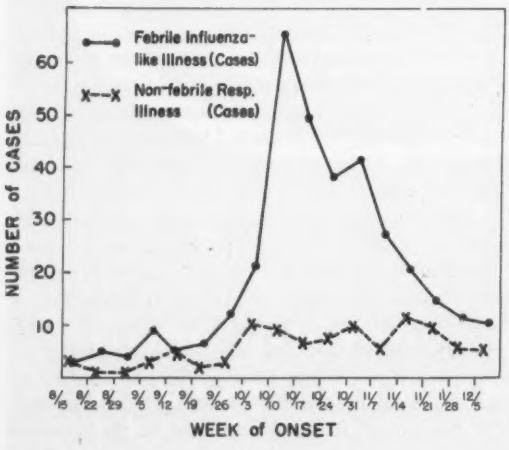
Results

There were 1,310 responses from the original 2,000 questionnaires distributed. The Asian influenza vaccine had been received by 640 of the 1,310 persons, and 651 persons were not vaccinated. An additional 19 persons had been vaccinated but became ill with respiratory illnesses less than 10 days after inoculation. Inasmuch as it has been shown that a significant increase in antibody usually does not occur in less than 10 days after inoculation, these 19 cases were excluded from consideration for evaluation of the vaccine effectiveness.

Recent respiratory illnesses were reported by 516 of the 1,310 persons answering the questionnaire, or an incidence of 39.4 per cent. A more severe illness with fever, myalgia, and respiratory symptoms occurred in 406 of the 516 individuals who were ill. The remaining 110 reported milder illnesses with respiratory symptoms but no fever. Clinically, most of the illnesses in the latter group would ordinarily have been classified as colds, except in 25 patients who also experienced gastrointestinal symptoms of diarrhea, frequently with nausea and vomiting.

The weekly incidences of the two groups of illnesses described above, based on the dates of onset, are plotted on the graph in Figure 1, where it may be observed that the peak of the epidemic of respiratory illnesses with fever was reached sharply during the second week of October 1957. This was about one week following the institution of influenza vaccination on a large scale at the Medical Center. It may be observed that there was some increase in the incidence of milder, non-febrile illnesses during the same period. However, the general contour of the curve is rather flat, and this observation leads us to believe that many, if not most, of these milder illnesses were probably caused by infectious agents other than Asian influenza virus.

Figure 1. WEEKLY INCIDENCE of RESPIRATORY ILLNESS at KUMC for 17 WEEKS, 8/15/57 to 12/5/57.



Among the 640 vaccinated individuals there were 90 cases of febrile respiratory illnesses, an incidence of 140.6 cases per 1,000. Each of these patients became ill at least 10 days after being vaccinated. Febrile respiratory illnesses were reported by 306 of the 651 unvaccinated persons, or an incidence of 470.0 cases per 1,000. Assuming that the incidence of febrile respiratory illnesses in this population during the stated period of observation was 470 cases per 1,000, then the expected number of such illnesses in the vaccinated group would have been 301 cases instead of the 90 cases actually reported. By using these figures the method of vaccination employed was estimated to have been 70 per cent effective against this group of febrile respiratory illnesses.

Ten individuals reported having two separate febrile illnesses occurring at least one month apart. Nine of the ten patients had not been vaccinated, and the tenth patient was vaccinated less than ten days prior to the onset of his second illness.

Mild, non-febrile respiratory and gastrointestinal illnesses were reported by 43 of the 640 vaccinated individuals, or 67.2 cases per 1,000. The incidence of such illness among the 651 unvaccinated persons was 58 cases, or 89.1 cases per 1,000. The differences between these two groups were so small that they did not appear to be statistically significant ($p=0.14$). This observation further leads us to believe that the majority of these non-febrile illnesses were not Asian influenza.

If all of the febrile and non-febrile illnesses are combined, then there were 133 cases of respiratory illness in the vaccinated group of 640 persons, or 207.8 cases per 1,000; and there were 364 cases of respiratory illness in the unvaccinated group of 651 persons, or 559.1 cases per 1,000. Accordingly, using the same method of calculation previously described, there should have been 358 cases of respiratory illness rather than the 133 cases reported in the vaccinated group; thus, the method of vaccination employed was estimated to have been 63 per cent effective against all types of acute respiratory illness.

Comment

This particular study is immediately vulnerable to two criticisms: (1) that the questionnaire method of collecting data is admittedly somewhat inaccurate, and (2) the quality of the statistics would have been greatly improved if it had been possible to obtain a larger number of virological studies. Despite these criticisms, it appears likely that the majority of cases of acute respiratory illness with fever were caused by the Asian influenza virus. It is also probable, for reasons already expressed, that the majority of the milder

TABLE 1

INCIDENCE OF RESPIRATORY ILLNESS AMONG EMPLOYEES AND STUDENTS OF UNIVERSITY OF KANSAS MEDICAL CENTER BY VACCINATED AND NON-VACCINATED GROUPS, FALL, 1957

Population Group	No. of Persons	Febrile Resp. Illnesses		Non-Febrile Resp. Illnesses		Total Resp. Illnesses	
		No. of Cases	Rate per 1000	No. of Cases	Rate per 1000	No. of Cases	Rate per 1000
Vaccinated	640	90	140.6	43	67.2	133	207.8
Non-Vaccinated	651	306	470.0	58	89.1	364	559.1
TOTAL	1291	396	306.7	101	78.2	497	384.9

illnesses without fever were not Asian influenza, although, in the absence of virological data, this belief can be accepted only as a clinical impression.

The Commission on Influenza of the Armed Forces Epidemiological Board[1] recently reported on the results of field studies executed by four different groups of investigators concerning the effectiveness of vaccines against Asian strains of influenza. The degree of protection in four groups inoculated with 200 C.C.A. units of Asian virus ranged from a low of 42 per cent to a high of 60 per cent.

Bell and his associates,[2] working with human volunteers, found a similar degree of protection in vaccinated persons challenged experimentally with a live virus (influenza A/Valley Forge/57) which is antigenically similar to the Asian strain virus.

In our own study, when the numbers of all respiratory illnesses, both febrile and non-febrile, were combined, the degree of protection afforded by the same method of vaccination approximates that of the aforementioned reports, 63 per cent. On the other hand, if only febrile respiratory illnesses are considered, the degree of protection in vaccinated persons appears to be somewhat better, 70 per cent.

Summary

This report describes a study of the effectiveness of vaccination with 200 C.C.A. units of the Asian virus in an epidemic of acute respiratory illnesses occurring among employees and students at the University of Kansas Medical Center during the fall of 1957.

Among 640 vaccinated persons the rate of febrile respiratory illness was 140.6 cases per 1,000, as opposed to a rate of 470 cases per 1,000 among 651 unvaccinated individuals. In this group of illnesses the method of vaccination was estimated to be 70 per cent effective.

When all respiratory illnesses, both febrile and non-febrile, were totaled, the incidence was 133 cases in the 640 vaccinated individuals, or 207.8 per 1,000; and, in the 651 unvaccinated persons, the incidence was 364 cases, or 559.1 per 1,000. Thus, the total over-all effectiveness of the vaccine against all respiratory illnesses was estimated to be 63 per cent.

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Life affords no higher pleasure than that of surmounting difficulties, passing from one step of success to another; forming new wishes and seeing them gratified. He that labors in any great or laudable undertaking has his fatigues first supported by hope and afterwards rewarded by joy.

—*Samuel Johnson*

Caustic Esophageal Stricture

A Review of the Literature and a Suggested Plan of Treatment

R. E. BRIDWELL, M.D., ***Kansas City***

Stricture of the esophagus, due to caustics, has long been a problem. The total number of cases is smaller today than 25 years ago, partly due to the decreased use of lye in the home and partly due to general public enlightenment on medical problems. However, it still remains a serious problem, and there is nothing more distressing to the patient, family, and physician than the prolonged treatment of an esophageal cripple.

The ultimate goal in this problem would be to prevent the accidents that lead to strictures, by education and legislation. Since this goal has not been accomplished at present, nor will it be reached in the forseeable future, it is important to search for new and better ways of dealing with chemical burns of the esophagus.

The seriousness of this problem can be appreciated by looking at some of the statistics of lye burns of the esophagus. Slightly more than 70 per cent of cases dealing with an esophagus burned by lye can be expected to develop strictures if untreated. It has been further stated that after the ingestion of lye, 58 per cent of patients experience severe dysphagia within one month, 80 per cent within two months, and, by the time eight months have gone by, 99 per cent of the ones who are going to develop strictures have done so. However, one must bear in mind that once the esophagus has been burned, a stricture may develop any time in the life of the patient. Stothers described the development of sudden acute impaction as long as 40 years after the accident.

The severity of the lesion depends on several variable factors. Among these are the nature of the agent injected, the quantity, the concentration, and the time of contact of the agent with esophageal mucosa.

Daly,[2] in a recent article, reports that a wide variety of agents with lethal possibilities had been swallowed. Among these were lye, Drano, washing soda, phenol, Lysol, formaldehyde, ammonia, smelling salts, Clorox, sulfuric acid, nitric acid, lactic acid, acetic acid, potassium permanganate, bichloride of mercury, and iodine. I would like to add Clinitest tablets to this list. I will have more to say later about this.

The chemical nature of the agent ingested has a great deal to do with the prognosis of the individual case. For example, alkalies have the ability to penetrate to the deeper layers of the esophagus because they destroy living tissue by a liquefying necrosis. On the other hand, acids produce a coagulative type of necrosis of the superficial layer of the esophagus, thus preventing the corrosive agent from reaching the underlying tissues by eschar formation. It is interesting to note that Lysol is frequently taken by individuals attempting suicide, but because of its chemical nature it produces only a superficial esophagitis. Healing of Lysol esophagitis is rapid and this agent seldom produces stricture formation. Iodine usually produces extensive superficial burns of the esophagus, but, again, most of these heal without stricture formation in the esophagus.

Since a stricture may develop at any time in the life of a patient whose esophagus has been burned by lye, it is important that the best known therapy be utilized. More than 70 per cent of such patients, if untreated, can be expected to develop strictures. An outline of therapy is presented.

Another factor that must be considered in the severity of the lesion is the concentration of the offending agent. The more concentrated the agent, the greater the severity of the injury, both in depth of penetration and extent. The experimental studies of Krey, as reported by Daly,[2] show there is a definite quantitative relationship between the depth of penetration of lye and its concentration, as follows:

Normal (3.8 per cent) solution of sodium hydroxide with a 10-second contact with esophageal mucosa produced necrosis of the mucosa and submucosa and involved some fibers in the inner longitudinal layer in rabbits.

3N (10.7 per cent) solution of sodium hydroxide produced necrosis extending to the circular muscle.

5N (16.4 per cent) sodium hydroxide produced necrosis extending to the outer longitudinal muscle.

7N (22.5 per cent) sodium hydroxide produced necrosis extending throughout the whole esophageal

wall and also affected, to some extent, the periesophageal tissues.

These figures take on a special meaning when one realizes that caustic compounds commonly found in the home contain from 10 to 50 per cent lye. It is not inconceivable that a concentrated caustic compound taken into the mouth and immediately expectorated could mix with the saliva to form a one normal solution of sodium hydroxide. If this were then swallowed, a burn could result that would produce necrosis of the mucosa and submucosa.

The quantity of the substance ingested is also to be considered in the severity of the lesion produced. This is proved by the fact that those individuals attempting suicide ingest a greater quantity of corrosive agent than those who ingest it accidentally and consequently have a greater degree of injury that often will involve the stomach and duodenum.

Various histologic changes take place once a corrosive agent has been in contact with esophageal mucosa. Belinoff has studied these changes in a dog's esophagus burned experimentally. He has divided the process into four stages; necrosis, ulceration, granulation, and scar formation.

In the initial phase there is a zone of intense inflammatory reaction surrounding the area of necrosis. The necrotic tissue sloughs, and this leaves an ulcerating surface beneath. This acute phase usually lasts about two weeks. By the end of one week the esophagus is thick and semirigid because of inflammatory edema. New blood vessels are beginning to form, and a fibroblastic process is initiated. After about 14 days the defects in the sloughing areas are filled in with granulation tissue. Some time in the third week the collagen fibers begin to contract, causing narrowing of the esophagus. Adhesions form between the granulating areas, and thus the scarring process begins to coalesce. After several months healing is accomplished by replacement of the muscularis mucosa and muscular coats with dense fibrous tissue. The epithelium that now covers this area of scar tissue is no longer normal squamous epithelium but is low stratified squamous epithelium, devoid of glands.

A patient who has swallowed a corrosive substance presents a diagnostic problem. First, is there an esophageal burn? It is important to keep in mind that there is no exact correlation between the condition of the mouth and pharynx and the extent of injury to the esophagus. This was brought out in an article by Daly who states: "A patient may have severe burns of the mouth and pharynx, without having any burns in the esophagus. Conversely, and much more important, burns may be absent in the mouth and pharynx and yet a severe burn of the esophagus be present."

There are two main methods of examination open to the physician to determine the condition of the esophagus. One is x-ray study of the esophagus with barium and the other is esophagoscopy. The first method is of doubtful value in the acute stage before the formation of stricture, but it is a useful method to determine the value of a given treatment once the stricture has developed. Therefore, it boils down to this simple fact: one must do an esophagoscopy to ascertain if there has been esophageal damage.

Esophagoscopy should be done within the first 48 hours after the accident. The "wait and see" doctrine still found in many modern medical texts is deplorable in the light of the modern instrumentation that is available. While esophagoscopy is not without risk, the hazard can be kept to a minimum if the procedure is done carefully and if no attempt is made to insert the esophagoscope through a burned area. In small children and uncooperative adults the esophagoscopy should be done under a general anesthetic to avoid unnecessary trauma to the esophagus.

Before discussing the method of treatment in these cases I would like to present reports of two patients with esophageal burn who were treated at the University of Kansas Medical Center. Then I would like to outline a regimen of treatment that seems reasonable, in the light of modern medical practices, once the diagnosis of esophageal burn has been suspected.

Case 1, Mrs. C. G., a 65-year-old colored housewife, was first seen in the emergency room on November 8, 1955, with the chief complaint of "took some poison."

The present illness began about ten minutes prior to her admittance to the emergency room. She had awakened in the morning with a headache and had taken a Clinitest tablet instead of an aspirin.

Past history revealed that the patient had been known to be diabetic for two years and was on a daily dose of 40 units of NPH insulin. Further questioning revealed that she had suffered from hypertension for several years prior to the diagnosis of diabetes.

When admitted to the emergency room the patient was vomiting and complaining of moderate burning pain in her chest. She was immediately given five glasses of vinegar water and two ounces of olive oil orally. The patient continued to vomit and brought up most of that which she had just taken. The burning pain subsided in a few minutes and she was released after being given another ounce of olive oil.

The patient was again seen in the emergency room three days later, at which time she complained of a lump-like sensation just to the right of the lower sternum. Questioning revealed that she had vomited a small amount of blood in the evening of the day she

was first seen. She had no dysphagia. She was referred to the Ear, Nose, and Throat Section for evaluation.

The patient was immediately hospitalized and esophagoscopy carried out under topical anesthesia. No buccal or hypopharyngeal burns were encountered, but she did have a severe annular burn 26 cm. below the incisor teeth.

When she was admitted to the hospital her blood pressure was 200/120, pulse 80 and regular. She was a moderately obese colored woman. The physical examination was not unusual.

She was placed on a 1,500-calorie liquid diet and given 40 units of NPH insulin each morning. She was started on penicillin, 600,000 units twice daily, and 25 mgm. of cortisone, orally, each four hours. While on cortisone therapy she had blood sugars twice daily and urine sugars four times per day. She maintained adequate diabetic control on the original 40 units of NPH daily. Cortisone and penicillin were continued for ten days.

On the second hospital day the patient was started on prophylactic esophageal bougienage with a 32 F. mercury laden Hurst bougie. The esophagus was dilated twice weekly while she was in the hospital and for the first month following her dismissal. The 32 F. mercury laden bougie passed with ease until one month after the ingestion of the Clinitest tablet, when a definite resistance could be felt 26 cm. below the incisor teeth. On several occasions a smaller bougie had to be passed before the 32 F. would pass.

In the second month of therapy bougienage was carried out once per week and in the third month every other week. The interval between dilations was gradually increased until the last interval was six months. At this time the patient had no dysphagia and no sign of stricture.

The patient had only minimal dysphagia during the first part of therapy and has been maintained on a regular diet since ten days after the accident.

Case 2, Mrs. M. W., a 56-year-old housewife, was first seen, as an emergency patient, the evening of November 12, 1955, with the chief complaint of "can't swallow."

The history of the present illness began one month before. She stated that she had inadvertently swallowed two Clinitest tablets instead of two aspirin tablets. She had almost immediate onset of burning pain in the chest and vomiting. She was seen by her family physician and was given vinegar water.

The family history was essentially negative. Past history revealed no previous hospitalizations or serious illness. It is interesting to note that this patient was not diabetic; it was her husband who was using the Clinitest tablets.

When admitted to the hospital the patient was suffering from complete esophageal obstruction. She was unable to swallow her saliva following the ingestion of a bolus of meat at lunch time. Esophagoscopy revealed an obstructive bolus of meat 30 cm. below the incisor teeth. After removal of the bolus of food, an almost complete stricture of the esophagus at this same area was found. Dilation of the esophagus was then carried out up to 36 F. The patient was then able to tolerate a liquid diet, but she stated that soft foods went down so slowly that she was afraid to eat them.

For the next several weeks the patient was in and out of the hospital numerous times for esophagoscopy and dilation. After six to eight weeks it was possible to pass a 32 F. mercury laden bougie, and she became an out-patient. She tolerated the dilations poorly and was nauseated and complained of abdominal pain, although she was on a liquid and soft diet most of the time. Since the patient lived out of town, the dilations with the mercury laden bougie have been carried out by her family doctor.

The treatment of caustic esophageal stricture by dilation usually spans many years, and many patients require dilation at varying intervals throughout their lives.

In recent years the use of cortisone has been advocated for the prevention of esophageal stricture after a caustic burn of the esophagus. Norman Rosenberg et al.[8,9] report experimental use of cortisone in the prevention of strictures in the rabbit esophagus after lye injury. They feel that cortisone is a potent inhibitor of the tendency to scar tissue contracture in the rabbit esophagus. However, the amount of cortisone used to produce these results would be equivalent to 420 to 500 mgm. per day for a 70 Kg. human. The mortality rate among experimental animals on cortisone alone was high. The concurrent use of penicillin favorably influenced the mortality rate, making it better than the rate when cortisone alone was used. I think one must conclude that use of such large doses of cortisone in the human subject would be dangerous.

Alex Wisskopf,[1] in a recent paper, concludes that cortisone and Terramycin are useful in the prevention of stricture formation in experimental burns in a dog's esophagus. He states that no stricture developed in the treated group, but all control animals living after 35 days developed strictures.

In attempts at the University of Kansas Medical Center to repeat this and similar experiments we have met with such severe mortality rates among control animals that we cannot predict with any accuracy the number of strictures that could be reasonably expected.

In reviewing recent literature one finds several

reports of the use of cortisone and antibiotics in treating human esophageal burns.[5] Some patients developed strictures and some didn't while on cortisone therapy. Many of the cases that were reported as successful were not documented with esophagoscopy, and it is certainly possible that the patients had no esophageal injury. Other successful cases, on cortisone therapy, have been reported after the ingestion of chemical agents that are known to produce strictures only rarely.

It has been shown experimentally that cortisone inhibits the formation of granulation tissue and thus the amount of scarring reaction. Theoretically and experimentally cortisone and antibiotic therapy should be beneficial in corrosive burns of the esophagus, and probably they are beneficial. However, I feel that not enough is known about the action of these agents in burns of the human esophagus to state that they will unequivocally prevent stricture formation.

I would like to outline a course of action that seems practical and reasonable at the present state of knowledge, realizing that it will need revision as our knowledge increases.

1. The patient should be hospitalized for the first two weeks of care.

2. Sedation and narcotics should be given to control pain the first few days.

3. Antibiotic prophylaxis should include penicillin, 600,000 units twice daily, and streptomycin, 0.5 gm. twice daily, for adults. Other antibiotics should be used for penicillin-sensitive patients.

4. Esophagoscopy should be conducted within 48 hours after the initial injury to determine the presence and the site of injury.

5. Cortisone should be given by mouth, 300 mgm. the first day, 200 mgm. the second day, and 150 mgm. daily thereafter for 28 days with careful observation of the patient for any untoward reactions to the cortisone therapy. For children the average dose should be 25 mgm. of cortisone each eight hours.

6. Administration of 1.5 to 2.0 gm. of potassium chloride daily will help to offset any potassium deficiency that might result from cortisone therapy.

7. Prophylactic esophageal bougienage should begin on the fifth day from injury and be conducted twice weekly for the first month; once every other week during the third month; then once per month for the next six months. This regime carries the patient through the critical eight months during which 99 per cent of those who are going to develop stricture have done so.

8. A liquid diet should be administered during the first week; then it should be gradually changed to a soft and to a bland diet, as tolerated by the patient.

9. Laboratory tests should be performed as indicated by the clinical course.

10. A gradual reduction of cortisone dosage should be planned over several days with terminal administration of ACTH gel.

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Perhaps the most valuable result of all education is to make you do the thing you have to do, when it ought to be done, whether you like it or not; it is the first lesson that ought to be learned; and however early a man's training begins, it is probably the last lesson that he learns thoroughly.

—*Thomas H. Huxley*

PRESIDENT'S PAGE

DEAR DOCTOR:

Of particular note at the University of Kansas Medical Center is the high quality, practical usefulness, *and* the quantity and broad diversity of continuing postgraduate instruction. These courses add up to a surprising 1/16 of the total of all postgraduate courses offered in the 83 medical schools and many hospitals in the 48 states.

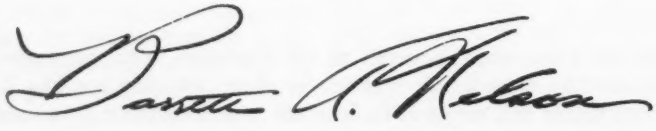
Over 50 per cent of Kansas doctors attended the postgraduate sessions last year, over 80 per cent at some time during the past five years. Most now pursue their continuation studies here, 79 per cent actually. Sixty-eight per cent are doing graduate work both here and elsewhere.

An amazing array of distinguished men in every field is imported for the graduate faculties, and our own faculty members contribute generously of their fine talents. The entire program is self-supporting, financed by the fees paid, excepting only the time contributed by members of the Medical Center staff. Many medical schools elsewhere are severely criticized for subsidization of graduate instruction costs, particularly by other groups, such as dentistry and the legal profession.

For those who feel they cannot leave their practices to attend courses at the Center, the Circuit Courses presented periodically at various cities over the state offer an additional opportunity to participate in the program. These meetings are uniformly well attended.

The school administration, in achieving these accomplishments, generously allots much credit to our Committees on Medical Schools and on Postgraduate Study for encouragement and providing stimulus to presentations, selection of suitable practical courses, study and analysis of answers to questionnaires, as well as in development of mutual enthusiasms. The result is of great benefit to medical practice and has contributed largely to the quality of the medical care we dispense.

Fraternally yours,



President

EDITORIAL COMMENT

School of Medicine Issue

Although the phrases which introduce the annual University of Kansas School of Medicine issue of the JOURNAL are becoming stereotyped, the pride that is felt by members of the Editorial Board is in no way dimmed. On the contrary, the Board's pleasure in this annual issue is increased because the occasion is repeated for the 12th time.

Appreciation is expressed to Dr. W. Clarke Wescoe, dean of the school, who contributed the lead article, and to Dr. Vernon E. Wilson, associate dean, who serves also as an associate editor of the JOURNAL. Credit for assembling all scientific material in this issue is due Dr. Wilson.

The Kansas Medical Society looks forward to publishing additional material from the Medical Center throughout the year and takes pleasure in the fine relationship which exists between the two organizations.

Evolution or Revolution

Revolutionary changes although sudden in onset are not sudden in inception. In each instance the violent nature of the change can be traced to the lack of sensitivity on the part of those who "have" to the needs and wants of those who "have not." In other words, evolutionary changes did not keep pace with the changing need, and the resultant increased pressures brought about a revolutionary move. A brief review of history will serve well to prove this point.

The responsibility for the formation of principles guiding the practice of medicine has traditionally been in the hands of dedicated and farsighted men. The initial effort to establish the American Medical Association was stimulated by the need to improve medical education and, thus, the care of the general public. The Flexner Report was but another self-disciplinary move on the part of practitioners and educators to keep the level of medical performance at a satisfactory level. More recently grievance committees, Blue Cross-Blue Shield, and other such endeavors have served as proof of the continued interest of organized medicine in the welfare of the public. With the rapid accumulation of new knowledge, the increasing demands made upon the physician for his time and intellectual efforts have tended to force him to focus more and more upon the initial acquisition of information to the neglect of the development of adequate evaluation of the results of his practice. In many instances time spent in training programs, certifications, and years of experience in medical practice have been confused with competence in handling medical problems. No man can possibly learn all about even a limited area of medicine regardless of the time and effort he may spend and it is therefore the knowledge of his own limitations, on the part of the physician, which becomes all important. Thus, experience and competence may well be totally dissociated depending upon the timing, quality, and type of experience. How then is competence to be determined?

The medical school can assume the responsibility of establishing standards and requirements for the granting of the M.D. degree; it can also assist in designing suitable graduate and postgraduate learning experiences. However, it cannot and should not become involved in assessing the excellence of medical practice. This is the rightful role of organized medicine and a privilege and responsibility which should be jealously guarded. Evolutionary changes have been made in this direction through hospital tissue committees, infection committees, grievance committees and other such organizations. None of these have been effectively used thus far to truly assess the practice of medicine in the community as a whole. Only the county, state, and national medical society can constructively and effectively enter into such a program. More importantly this cannot be a police action but must result from the intense desire on the part of the majority of the members to participate in a vigorous and continuing self-appraisal.

How is this to be accomplished? Certainly written examinations are not the answer. Very probably the techniques of using review committees at the county and state medical society level is the most available and efficient method. In the final analysis it is the quality of medical care given which should be used as a standard. A man's knowledge of his own limitations and of the available resources which can be used, both personnel and facilities, is the chief protection available to the patient. Whether or not the physician has done what the prudent practicing physician of his community would have done is the criterion used by the court of law.

If we in organized medicine do not rise to meet this growing need, other groups and organizations will do it for us—business concerns and governmental agencies have already taken some steps to establish arbitrary criteria for the hiring of physicians. These are based upon unsound and, in some instances, dangerous principles. The alarming growth of malpractice suits is but another warning sign of increasing pressure. Ideally our enlightened approach to evolutionary changes will continue to pace the wants and needs for health care. An enlightened public has given evidence that it will settle for no less. Hopefully, revolutionary methods with their inevitably destructive and wasteful mechanisms can be avoided. This can be guaranteed if physicians as a group will apply them-

selves to the diagnosis and treatment of this problem with the same vigor, thoroughness, and intelligent application they have used in treating their patients.—*Vernon E. Wilson, M.D., Associate Dean, University of Kansas School of Medicine.*

Reasons for Pride in Our School

It is no exaggeration to state that Kansas has one of the most outstanding medical schools of the nation. It is outstanding and unique in the meticulous, scientific selection of student applicants, in organization of highly developed courses presented in curricula of the most modern and "progressive" character, a faculty of distinguished teachers, both in the basic sciences and in clinical subjects, unique in its close liaison with active practitioners and in active co-operation with organized medicine.

Physicians in practice are most interested, naturally, in the postgraduate curricula. As recounted on the President's Page, this department is not only remarkable in its broad development, but, also, in its active and enthusiastic utilization by the profession.

The medical school now matriculates 105 students each year. Approximately 93 per cent are Kansans; 5 to 6 per cent are from Kansas City, Missouri (really more Kansan than Missourian), only 1 to 3 per cent from other areas. Indicative of the quality of the matriculants selected is the small number dropped from the rolls. For example, last year only three freshmen failed to produce acceptable work; two withdrew because of illness; two discovered they had embarked upon a profession for which they were not properly fitted. The number dropped in the second, third, and fourth years is practically nil.

Curricula are effectively planned, not only to provide sound, fundamental, scientific training, but are distinctively "progressive" in character. This is shown in the trend toward opportunities for the exceptional student to exploit his special talents or particularly intense interests.

Each senior medical student spends 4½ weeks at a state hospital for mental health, where he discovers the tremendous importance of this field, the great need for better understanding of mental health needs, and a comprehension of the value of properly organized, amply supported hospitals and programs for treatment of mental illness.

The preceptor program is, of course, the unique Kansas development with each senior student spending 4½ weeks in the office of a small town general practitioner. Dr. Mahlon Delp, brilliant professor of medicine, terms it "the best possible way of teaching a medical way of life." It is an excellent teaching program, presenting the practical aspects of medical practice, relationships with the private patient, with the family of the patient, and with the community. Students are pleased and interested in this period of training. No definite figures are available as to the number who are motivated to enter rural or small town practice, but their number is considerable. Before inception of the preceptor program only 33 per cent of graduates remained in Kansas. This rose fairly rapidly to 50 per cent, and has now reached 60 per cent, almost twice as many as formerly. During the past year, as instances, three graduates have established practices in Belleville and others in Haven, Quinter, Russell, and in other Kansas towns.

The preceptor program is arousing more and more national attention. It was a featured topic for discussion at national medical school meetings during the past few months.

It is entirely accepted that enormous credit is due our dynamic, resourceful, personable dean, Dr. W. Clarke Wescoe, and genial, capable, co-operative associate dean, Dr. Vernon E. Wilson. Both are well known to the profession by their friendly democratic participation in society activities, their open-mindedness toward our advisory groups, and their obvious desire to further the best interests of Kansas medicine. Dr. Wescoe was enthusiastically received by the Council on Medical Service of the A.M.A., where he already has obtained rapt attention for presentation of specific ideas and has become chairman of an important sub-committee. Dr. Wilson holds the office of assistant secretary of the Association of American Colleges, an important body where he plays an effective role in the reviewing and rendering of reports on the accreditation of medical schools.

Finally, the year by year growth in the physical plant is truly amazing, the fine modern buildings, expanding bed capacity, clinical laboratories, teaching areas, research facilities, the auditorium, and the attractive, comfortably usable postgraduate facilities. It, indeed, has become a metropolitan medical center to be proudly regarded with the finest anywhere.—*Barrett A. Nelson, M.D., President, Kansas Medical Society.*

Income Tax Guide Available

"The Federal Income Tax Guide for Physicians" is the title of a booklet published recently by the Law Department of the American Medical Association. It contains answers to some of the most perplexing tax problems, compiled from court decisions as well as rulings, regulations, and publications of the Internal Revenue Service. It has been designed to give physicians a better understanding of their rights and obligations under federal income tax laws.

The Law Department staff has only one word of advice: Do not consider this booklet as a *substitute* for the services of a personal tax advisor!

Maternal Death Study—Case History

Editor's Note. This is the third of a series of case reports prepared by the Committee on Maternal Welfare to illustrate the type of study made in each instance of maternal death in Kansas. The committee also records its decisions as to the most probable cause of death, the preventability or unpreventability of the death, and the major contributing factors.

The patient was a 32-year-old gravida 8, para 6, abortion 1 who reported for prenatal care in the seventh month of her last pregnancy. The record reveals no gross abnormalities, but the diet was considered below standard. The serological test was the only laboratory procedure performed. She had three subsequent visits prior to delivery.

About 3:30 p.m. four weeks before term, she slipped and fell on an icy walk. Pain in the back and side developed shortly and became increasingly severe, requiring hospitalization at 5:55 p.m. There was no evidence of labor at that time, temperature, pulse, respirations, and blood pressure being normal and x-rays of the back and lumbosacral spine revealing no abnormalities. No fetal heart tones were audible, however. She was given Demerol, 50 mgm., and blood was typed. The following events ensued:

7:00 p.m. True labor began.

7:25 p.m. Contractions hard, small amounts of bright red blood gushing from the vagina.

8:00 p.m. Patient taken to delivery room.

9:07 p.m. Cervix dilated 3-4 cm. Pitocin, 3 minims given.

9:27 p.m. Same. Pitocin, 5 minims given.

10:00 p.m. Note made that baby was dead. Patient to be returned to her room for blood transfusion.

10:45 p.m. Transfusion started (500 cc.).

12:45 a.m. Nitrous oxide anesthesia started in delivery room. Demerol, 50 mgm., given.

1:05 a.m. Delivery of stillborn, four-pound, two-ounce fetus.

1:15 a.m. Delivery of placenta with copious blood loss. Pitocin, one ampoule, and Ergotrate, one ampoule, given. Patient pale and cold. No pulse or blood pressure recorded.

1:20 a.m. Patient returned to room. Blood, 500 cc., started.

2:00 a.m. Ergotrate, one ampoule given. Thorazine, 25 mgm.. given.

2:40 a.m. Blood pressure 50/10. Koagamin, 3 cc., given intravenously.

2:45 a.m. Vasoxyl, 1 cc., given intramuscularly.

3:00 a.m. Blood, 500 cc., started.

3:20 a.m. Vasoxyl, 1 cc., and caffeine sodium benzoate, 2 cc., given.

4:10 a.m. Uterus noted to be firm. Strychnine, gr. 1/60, and sodium phenobarbital, gr. 3, given.

5:40 a.m. Respiration slow, pulse weak and rapid. Oxygen started; Coramine, one ampoule, given.

6:07 a.m. Patient pronounced dead.

The notations constitute all information recorded at the time. The physician remained in constant attendance but did not call for consultation.

COMMITTEE ACTION: The committee noted the failure to make a diagnosis and failure to take positive action. There is no record of blood pressure having been taken except on admission and at 2:40 a.m. There was no indication that the uterus was adequately checked to rule out a laceration or rupture. It was felt that Pitocin was used unwisely and excessively in view of the parity of the patient. In view of the good resources available, it was thought particularly unfortunate that consultation was not obtained.

The inadequate record prevented the committee from determining the principal factor in this death, but it was thought to have been ablation of the placenta or rupture of the uterus. The death was considered preventable with the error in judgment of the attending physician being the major contributing factor.

CLASSIFICATION: OBSTETRICAL DEATH; PREVENTABLE; ERROR OF PHYSICIAN; NO AUTOPSY.

Clinicopathological Conference

Abdominal Pain, Shock, Thrombocytopenia, and Rapid Enlargement of the Liver

Case Presentation

The patient under consideration today, a 45-year-old white woman, first entered KUMC at 1:00 a.m. on March 23, 1956, and died at 11:10 p.m. on March 24, 1956.

She had been perfectly well until January, 1956, when she had a sudden onset of squeezing substernal pain which radiated to the right shoulder and down the right arm. This pain was associated with dyspnea, and it lasted 36 hours. She was hospitalized for three days at that time. Afterwards, she was apparently well until two weeks before admission, when pain again developed in her right shoulder. This pain again lasted for 36 hours, and she was hospitalized for one week at that time.

On March 21, 1956, she had a periumbilical pain which radiated to the back and was associated with nausea and diarrhea. Her stools were yellow, had a foul odor, and floated on the water. On March 22, 1956, the abdominal pain was in the right upper quadrant, and she was nauseated and vomited throughout the day, but had no diarrhea.

A benign tumor was removed from the left breast in 1953; she had a pan-hysterectomy for fibroids in 1954. She was gravida VII, para VII.

The family history was non-contributory.

The patient was a well developed, well nourished white woman, slightly cyanotic and acutely ill. The systolic blood pressure was 60, and the diastolic pressure could not be estimated. The pulse was faint, and the rate was 120. The respiratory rate was 25. Her lips were cyanotic; the conjunctivae and oral mucosa were pale. The tongue was dry; the eyes, ears, and nose were normal. The lungs were clear. The heart tones were faint, and no murmurs were heard. The spleen and liver were not felt on admission, but later the liver was palpated about 10 cm. below the right costal margin. She had generalized rebound tenderness in the right lower quadrant. There was no rigidity, and the extremities and neurological examination were normal.

The specific gravity of the urine was 1.024 with acid reaction, 3 plus albumin, and negative sugar. The specimen was loaded with amorphous material and numerous epithelial cells, and there were 15 to 20 white blood cells per low power field. The red count was 5,490,000 with 15.2 gm. hemoglobin. The white count was 19,000 with 15 polymorphonuclears, 12 filamented, 76 lymphocytes, 1 basophil, and 8 monocytes. There were many immature lymphocytes and 15 nucleated red blood cells per 100 white blood cells. The platelet count was 16,000. Serum calcium was 3.8 mEq/L; phosphorous, 3.5 mEq; chlorides, 110 mEq. The nonprotein nitrogen was 47 mg. per cent; sugar, 290 mg. per cent; amylase, 150 to 200 units per 100 ml.

The foot of her bed was elevated, she was given oxygen and kept warm. A Levine tube was attached to suction. She was given intravenous and intramuscular hydrocortisone sodium succinate (totaling 600 mg.) and intravenous glucose and saline. In spite of these measures she remained in progressively deeper shock throughout her hospitalization. She died quietly on March 24, 1956.

Dr. Mahlon Delp (moderator): Are there any questions for Dr. Schmaus?

Robert E. Wildin (fourth year medical student):* Was there any diabetes in the family?

Dr. John Schmaus (resident in medicine): No, there was not.

Keith Woolpert (fourth year medical student): Were any other studies made during her previous hospitalizations?

Dr. Schmaus: A gallbladder study was done, and it was normal.

Mr. Wildin: Was there any history of alcohol ingestion?

Dr. Schmaus: No.

Mr. Wildin: Whooping cough?

Dr. Schmaus: No.

Mr. Woolpert: Was there any abdominal distention, hematemesis, or hemoptysis?

Dr. Schmaus: The abdomen was only slightly distended. There was no hematemesis or hemoptysis.

Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center, as well as by the third and fourth year classes of medical students.

* Though a medical student in March, 1957, when this conference occurred, he, like the others referred to as students, received the M.D. degree in June, 1957.

Robert L. Wilson (fourth year medical student): Were any bowel sounds elicited?

Dr. Max Allen (internist): There was no abnormality.

Paul J. Uhlig (fourth year medical student): Was there any evidence of subcutaneous bleeding, ecchymoses, or petechiae?

Dr. Schmaus: Small petechiae developed while the tourniquet was in place for the "cutdown."

Roger D. Warren (fourth year medical student): What was the temperature course while she was in the hospital?

Dr. Schmaus: On the day of admission the rectal temperature was 99.6 degrees at 7:00 a.m., but it was up to 101 degrees at 3:00 p.m. and at 7:00 p.m. On March 24 the temperature was 99.2 degrees, and just before death it was 98.4 degrees.

Mr. Warren: Did she receive intravenous glucose before the blood sugar was drawn?

Dr. Schmaus: Yes.

Mr. Wilson: Were there any lupus erythematosus cells?

Dr. Schmaus: We did not look for them.

Mr. Warren: Will you describe the Levine tube drainage?

Dr. Schmaus: It became slightly bloody.

Mr. Uhlig: Was there a history of hypertension?

Dr. Schmaus: No.

Mr. Wilson: What was the blood count on admission?

Dr. Schmaus: On admission the hemoglobin was 15 gm. and the white count was 1,800 with 8 polymorphonuclears, 4 filamented, 28 lymphocytes, 4 monocytes, and 60 young blast forms with 10 nucleated red cells per 100 white cells.

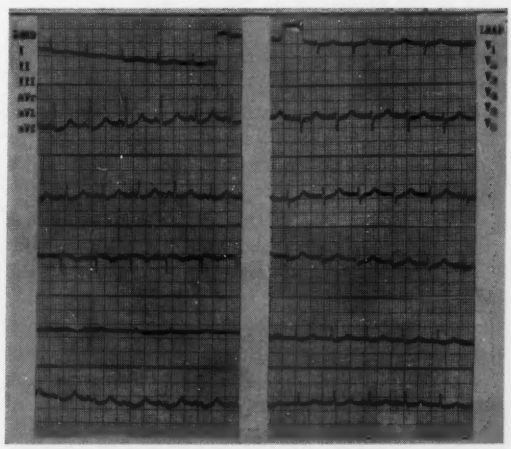


Figure 1. First electrocardiogram taken one day before admission.

Mr. Warren: Will you describe the patient's appearance on the day after admission?

Dr. Schmaus: Her appearance was essentially the same throughout her hospitalization.

Mr. Wildin: Was there any history of melena or steatorrhea before March 21?

Dr. Schmaus: No, there was not.

Mr. Woolpert: Were vasopressor drugs used during her hospital course?

Dr. Schmaus: She received levarterenol (Levophed) in an attempt to maintain her pressure, but it did not help.

Dr. Joaquin Lopez (resident in pathology): Were there any symptoms of bleeding, bruises, or hematemesis?

Dr. Schmaus: There were purpuric areas over her arms during her hospitalization here.

Dr. Delp: May we see the electrocardiograms, please?

Mr. Woolpert: The first electrocardiogram was taken on March 22, one day before her admission to this hospital (Figure 1). There is a normal sinus rhythm with a rate of approximately 110. The P-R intervals are within normal limits. There is a suggestion of peaking of the T waves. The QRS and T vectors appear to be within normal limits.

The next tracing, taken on March 23, the first day of admission, shows a slight increase in the rate. There is still a normal sinus rhythm.

Another tracing taken on March 24, 1956, shows a rate of approximately 110. There is peaking of the T waves and a suggestion of depression of the S-T segments in V_3, V_4 and V_5. This may suggest myocardial ischemia. This tracing may be compatible with hyperpotassemia and hypocalcemia.

Dr. Delp: Dr. Lin, do you have any comments?

Dr. T. K. Lin (cardiologist): The first tracing shows a fairly normal QT interval; however, if compared with a later tracing the QT interval is a little shorter. I do not believe one can diagnose hyperpotassemia.

Dr. Delp: Do you think the T waves are peaked?

Dr. Lin: They may be slightly peaked.

Dr. Delp: May we see the portable film, please?

Mr. Wilson: The portable film was taken on the day of admission with the patient in the lateral position (Figure 2). One can see the Levine tube and a bubble of air which indicates that she was lying on the left side. There is an area along the inside of the peritoneum which may be air. I do not believe that this represents abnormal intestinal obstruction.

Dr. Delp: Dr. Tice, do you have any comment?

Dr. Galen Tice (radiologist): This film was taken to determine whether there was any free air in the peritoneal cavity. She could not stand up, so the film

was taken with the patient lying on her right side. No air was seen in the peritoneal cavity.

Dr. Delp: May we have the discussion, Mr. Wildin?

Differential Discussion

Mr. Wildin: This case presents many diverse clinical and laboratory findings. I would like to base my discussion only on those diseases which we seriously considered in attempting to explain our patient's course.

In January, 1956, she entered the hospital with a history that was suggestive of myocardial infarction. For three days she had had periumbilical pain radiating to the back, nausea and vomiting, diarrhea and steatorrhea. Two months later she entered this hospital in a state of dehydration and shock. Laboratory findings included albuminuria, hypocalcemia, hyperglycemia and slight azotemia, and suggested acute pancreatitis. However, there were nucleated red blood cells in the peripheral blood, an absolute lymphocytosis with immature and blast forms, slight myelocytosis, neutropenia, and a marked thrombocytopenia. Acute pancreatitis alone cannot explain these findings.

There are three types of diseases that may offer an explanation: chronic lymphatic leukemia, acute lymphatic leukemia, and malignant lymphoma. Chronic lymphatic leukemia can be ruled out because our patient did not have a protracted course, and there was no peripheral lymphadenopathy, severe anemia, or splenomegaly.

Factors against the diagnosis of acute leukemia are the absence of palpable lymph nodes, splenomegaly, anemia, and tenderness over the sternum. These are variable findings in this disease, and for that reason one cannot dismiss it completely.

Figure 2. Portable film taken on day of admission showing the patient in the lateral position.

Malignant lymphomas without peripheral adenopathy can occur in the mediastinum, in the abdomen, or both. A mediastinal lymphoma either by rapid enlargement or by infiltration of the heart or pericardium with pericarditis and coronary occlusion could have explained her condition in January. This is unlikely, however, because the electrocardiogram shows no evidence of an old myocardial infarction.

There are no chest x-rays to indicate whether mediastinal nodes were present. Lymphomas in the abdomen may be inside or outside the bowel wall. Lymphomas found in the bowel are usually in the cecum or the small intestine. Small bowel lymphomas frequently cause a sprue-like syndrome which lasts for several weeks and results in death from intussusception, intestinal obstruction, or perforation and peritonitis. This may be a possible explanation of our patient's illness, but the course of this disease is usually more prolonged.

I do not believe that steatorrhea for three days can explain the hypocalcemia in our patient, and I cannot explain it without postulating that she had acute pancreatitis. As a consequence, I am faced with the problem of relating acute pancreatitis and lymphoma. Perhaps she had both diseases unrelated to each other, but we might postulate invasion of the pancreas and its ducts by a lymphoma causing obstruction and disruption of the pancreatic ductal system and leading to acute pancreatitis. Occlusion of the blood supply or venous drainage of the pancreas by infiltration or by thrombosis might also occur.

Right heart failure rarely causes such rapid and severe enlargement of the liver, as this patient had. A better explanation is that she terminally had thrombosis of the hepatic veins, the so-called Budd-Chiari syndrome which occurs with lymphoma, leukemia and inflammatory disease in the upper abdomen, such as pancreatitis. Shock and dehydration also predispose to thrombosis of the hepatic vein. Our patient could have had acute leukemia with decreased resistance to infection, infectious pancreatitis, shock, and finally thrombosis of the hepatic veins.

In summary, I believe that our patient either had acute leukemia or an abdominal lymphoma with terminal leukemia and terminal thrombosis of the hepatic veins. I believe that she died of irreversible shock which was secondary to pancreatitis and which led to anoxia of all the viscera, particularly the liver and adrenals. There may also have been infiltration of the adrenals with leukemic or lymphoma cells, infarction of the adrenals from shock or thrombosis, bleeding into the peritoneal cavity as a result of the thrombocytopenia, hepatic vein thrombosis, and car-

diac dilatation due to a combination of hypocalcemia and hyperpotassemia.

Clinical Discussion

Dr. Delp: Thank you. Mr. Warren, how do you explain the persistence of the shock? We know it existed throughout her final hospitalization.

Mr. Warren: The initial clinical examination gave evidence of acute pancreatitis or other acute abdominal catastrophe.

Dr. Delp: Mr. Woolpert, do you believe that this patient could have had a Waterhouse-Friderichsen syndrome?

Mr. Woolpert: Yes.

Dr. Delp: Can you choose just one cause for the shock?

Mr. Wildin: Acute pancreatitis.

Dr. Delp: Mr. Warren, how do you explain the enlargement of the liver which occurred during the patient's hospitalization here? Several competent examiners found that the liver was not enlarged when she was admitted, and an equally competent consultant found the liver 10 to 15 cm. below the right costal margin.

Mr. Warren: There is rapid enlargement of the liver in the Budd-Chiari syndrome. It could also explain the shoulder pain and generalized abdominal tenderness.

Dr. Delp: Are you referring to her chest pain of several weeks ago?

Mr. Warren: No, that could not be the result of her terminal disorder.

Dr. Delp: Mr. Wilson?

Mr. Wilson: I believe her liver enlarged rapidly because of thrombosis of the hepatic veins.

Dr. Delp: Mr. Woolpert?

Mr. Woolpert: Experimentally, fat infiltration has been reported during acute pancreatitis, but this has not been proved in human subjects. My best explanation is the Budd-Chiari syndrome.

Dr. Delp: How many such cases have been reported?

Mr. Wilson: I think that about 57 have been recorded since 1884.

Dr. Delp: Is the platelet count satisfactory to you, Mr. Warren?

Mr. Warren: No, it is not.

Dr. Delp: What was the report, Dr. Schmaus?

Dr. Schmaus: The platelet count was 16,000.

Dr. Delp: What do you think about that, Mr. Warren?

Mr. Warren: Thrombocytopenia with nucleated red cells and immature forms of all series of myeloid cells would suggest a generalized invasion of the bone marrow. That disturbs me somewhat, however, because she did not have peripheral lymphadenopathy.

Dr. Delp: Is this an extremely low platelet count?

Mr. Warren: It certainly is.

Dr. Delp: To what do you attribute this low platelet count?

Mr. Warren: I attribute it to megakaryocyte destruction.

Dr. Delp: Mr. Uhlig?

Mr. Uhlig: I attribute it to leukemia.

Dr. Delp: Mr. Wilson?

Mr. Wilson: There is no history of any toxic products which would cause a drastic reduction so quickly. She did not have anemia, but she did have thrombocytopenia. I believe that leukemia is the explanation.

Dr. Delp: It did happen awfully quickly, did it not? Do you feel a little insecure about it, Mr. Woolpert?

Mr. Woolpert: Cases of thrombosis and of the Budd-Chiari syndrome have been reported in which there was a decrease in platelets, but I do not believe the thrombocytopenia has ever been extreme.

Dr. Delp: Dr. Allen, may we have your comments?

Dr. Max Allen (internist): The findings were not too atypical. There were several factors that did not relate to the diagnosis of acute pancreatitis. The steatorrhea, the low serum calcium, and the shock, which is quite prominent in acute hemorrhagic pancreatitis, all relate to the disease. There was, on the other hand, the lack of ileus which is common in acute pancreatitis. The single most significant feature was the rapidly enlarging liver. My only explanation was that the patient was in a shock-like state with reduced cardiac output. We were replacing fluids rather rapidly. We attributed the shock to adrenal failure, due either to hemorrhage of the adrenals or to stress exhaustion of the adrenals. We concluded that this was an acutely developing situation, an acute leukemia, which had not had time to develop enlarged peripheral lymph glands and splenomegaly. My final impression was acute leukemia with hemorrhagic destruction of the adrenal glands and irreversible shock.

Dr. Delp: Dr. Wilson, what was your opinion of the platelet count?

Dr. Sloan Wilson (hematologist): In fulminating toxic states the blood picture may suggest acute leukemia. It is difficult to determine whether this patient had a toxic state or acute leukemia. I made a diagnosis of acute leukemia after having seen the smears containing a fulminating type of immature cell.

Dr. Delp: Why did the platelets disappear? Were they lysed, or are you postulating that they were not made?

Dr. Wilson: There are many ways in which platelets may disappear from the peripheral blood stream. The most common way, of course, is that seen in idiopathic thrombocytopenia in which there is a sudden lysis of platelets.

Dr. Delp: Dr. Schmaus, what was your diagnosis?

Dr. Schmaus: At first my diagnosis was acute pancreatitis, but I later changed it to acute leukemia.

Dr. Delp: Dr. Mantz, will you please present the pathology?

Pathological Report

Dr. Frank A. Mantz, Jr. (pathologist): It is of extreme gratification to me to confirm many of the statements the students have made here today. However, I must state at the outset that the pancreas was examined in great detail, and no evidence of necrosis or inflammation was found. The body for examination was that of a well developed, well nourished white female who appeared to be the stated age of 45 years. In keeping with the thrombocytopenia which was described, there were multiple petechiae throughout the skin, pleura, pericardium, and the urinary tract. No lymphadenopathy was discovered.

Figure 3. Bone marrow. Abnormal myeloid proliferation fibrosis and osteogenesis occurring in the same field.

Figure 4. Hepatosplenomegaly with focal necrosis in the liver and myeloid metaplasia of spleen.

Examination of many organs including the brain disclosed within the capillaries large numbers of highly immature cells both of the erythroid and leukocytic variety. The exact identification of the leukocytes is not possible in the tissues. The bone marrow was exceedingly firm and red to mottled gray in color. Histologic study disclosed that the marrow spaces were rather heavily infiltrated with small lymphocytoid cells. This showed a distinctly invasive behaviour by infiltrating beneath the periosteum in some areas and elevating it. The nature of the reaction in the bone marrow is exceedingly interesting. We noted that there was rather extensive distortion of architecture and that large numbers of megakaryocytes, cells of the erythroid series and reticuloendothelial elements were scattered throughout. The predominant cells, however, were distinctly lymphocytoid but, when studied with Giemsa stain, were found to contain granules and were apparently myeloblasts or promyelocytes.

We may conclude, therefore, that, in addition to diffuse proliferation of bone marrow, there was marked immaturity of the myeloid series. A reticulum stain of the bone marrow revealed an abundant depo-

Figure 5. Thrombus in hepatic vein with central necrosis of adjacent lobule.

sition of this material in a highly irregular and non-architectural pattern. This reflected the large number of reticular elements throughout the bone marrow, as well as the character of the neoplastic proliferation with which we deal. The bone marrow also showed areas of scarring containing tiny foci of new bone formation (Figure 3).

A similar process was encountered within the liver where there were small clusters of proliferating reticuloendothelial cells, scattered megakaryocytes, and numerous tiny islands of myeloid activity as well as erythrocyte production. Many of these were found within the portal areas.

The spleen also exhibited this phenomenon with relatively large numbers of megakaryocytes, many blast cells, and occasional islands of erythroid activity.

All of you recognize this, I am sure, as a diffuse myeloproliferative disorder, and it is not my intention at this time to belabor the argument whether or not the myeloproliferative states represent a variation of leukemia or a different entity. I wish, however, to point out that Damashek[1] has emphasized that the bone marrow may, under certain conditions, react en masse not only with the hematopoietic elements alone, but also with the stromal elements contributing to the proliferative or neoplastic process. Various results, apparently, may occur. One of these is typical chronic myelogenous leukemia. Similar alterations may also be seen in the recovery of the bone marrow from intoxications such as irradiation and in the metamorphosis of polycythemia vera to a leukemic state. It is most unusual, however, to see acute myelogenous leukemia complicating the myeloproliferative disorders. These are popularly known as agnogenic myeloid metaplasia, but in our laboratory they are classified as panmyelosis. In any event, there appears to be multiple differentiation of the mesenchymal cells into hematopoietic elements, fibroblasts, and osteoblasts, so that marked variation in the picture may occur.

The liver was the site of a rather amazing pathological alteration (Figure 4). It was markedly enlarged and weighed 2,300 gm. It was exceedingly soft and mushy and extended 8 to 10 cm. below the right costal margin. The cut surface was striking in that it showed numerous small focal areas of hemorrhage in the central portions of the lobules. Associated with this were foci of necrosis tending to become confluent to produce irregular masses of yellow discoloration. The

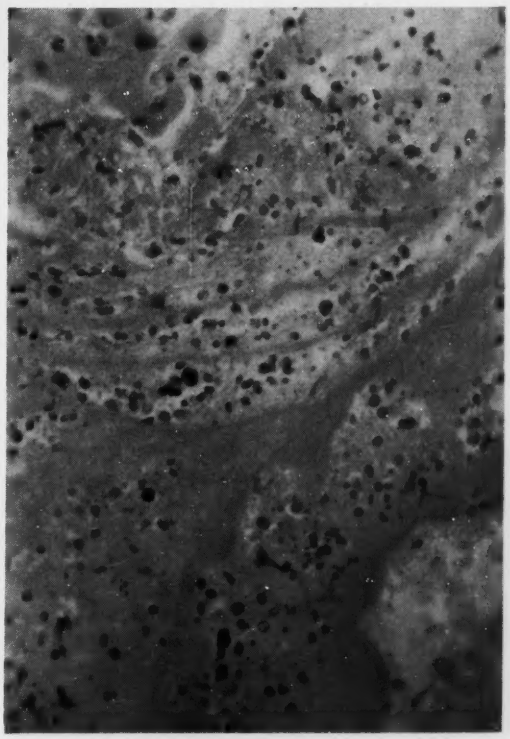


Figure 6. Thrombus in hepatic vein containing myeloid cells and megakaryocytes.

hepatic veins on the cut surface were markedly dilated and many of them grossly contained distinct thrombi.

Sections of the liver confirmed the presence of thrombi throughout the hepatic veins (Figure 5). The majority of these were fairly recent, but other thrombi were distinctly old, having undergone organization. I believe that the patient's initial complaints were related to these more remote lesions.

The mechanism of venous thrombosis appears related to the myeloproliferative disorder since many of the involved vessels contained myeloid cells within their walls. Many of the thrombi likewise contained megakaryocytes in addition to the leukemic type of cells (Figure 6). It seems probable that these elements may have precipitated thrombosis. Furthermore, the granularity of the thrombi suggested the possibility that platelets themselves may have agglutinated into solid masses explaining in part the reduced platelet count which was noted clinically.

As might be expected, there was extensive focal necrosis within the lobules in the manner of incomplete infarcts. The sinusoids were markedly dilated and many focal hemorrhages were seen. Disse's spaces were rather uniformly dilated, suggesting accumulations of lymph, a factor which may possibly explain the ascites which is so frequently seen with hepatic vein thrombosis. Six hundred ml. of ascitic fluid had collected in the abdomen.

I am sure that you are now aware that this is an example of the Budd-Chiari syndrome which was first described in 1846 by Budd and later, in 1894, further described by Chiari. It is a rare clinical entity although the pathologist recognizes it a great deal more frequently than does the clinician since hepatic vein thrombosis occurs as an event in a number of illnesses. On reviewing the literature one finds that the hematologic disorders are not uncommon. Among them polycythemia appeared to rate first, sickle cell anemia also has been described, and there have been a few cases in which the syndrome has occurred with leukemia.

The spleen was the site of extensive congestion. It weighed 600 gm., and histologically it presented the appearance of hemorrhagic infarction with almost all of the splenic parenchyma gone. All that remained were the myeloproliferative centers with which the spleen was so extensively filled.

The gastrointestinal tract showed evidence of portal hypertension in the form of massive hyperemia and numerous petechial hemorrhages. There was evidence that early collaterals had been established in the esophagus where there was striking dilation of the esophageal veins associated with hemorrhage into the surrounding tissue. This section was obtained at the site of a small erosion and presumably may have contributed to the bloody return from the Levine tube.

This patient was in considerable shock, and it is significant that she put out only 130 ml. of urine during hospital observation. Examination of the kidneys showed remarkable changes. Their weight together was 300 gm.; they were exceedingly pale, and at the outer part of the cortex the pallor was more evident than that seen in the inner aspect. Within the outer two-thirds of the renal cortex of both was a symmetrical zone of marked necrosis and ischemia as opposed to the inner one-third of the cortex where the parenchyma was not only well preserved but relatively hyperemic. This is bilateral symmetrical cortical necrosis of the kidneys in an early phase. It has been reported in pregnancy and in acute hepatic necrosis. It has been postulated that possibly some nephrotoxin may be elaborated in the liver to initiate this lesion. It has been pointed out that the bilateral necrosis of the kidney has a tendency to appear in shock due to any cause, and it has been reproduced in experimental animals by producing shock. We feel that the possibility of a pressor substance released from a necrotic liver may have accounted for intractable shock and facilitated these changes in the kidney which in most cases are irreversible and lead to death.

To summarize, I believe that this is a case of panmyelosis in which acute myelogenous leukemia was superimposed. As a result of the myeloproliferative disorder, thrombosis of the hepatic veins ensued resulting in shock and ultimately bilateral symmetrical cortical necrosis of the kidney.

Dr. Delp: Thank you, Dr. Mantz. In the future, shock-like states and a rapidly increasing size of the liver will suggest to all of us the possibility of hepatic vein thrombosis.

Pathological Anatomical Diagnosis

Primary

Chronic panmyelosis with myelofibrosis, osteosclerosis, and acute myelogenous leukemia.

Multiple old and recent thrombi, sometimes incorporating megakaryocytes, of small hepatic veins and central veins of liver consistent with the Budd-Chiari syndrome.

Focal lipoid depletion of adrenals, advanced.

Bilateral cortical necrosis of the kidney.

References

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2. Linman, J. W., and Bethell, F. H.: Agnogenic myeloid metaplasia, Am. J. Med. 22:107, 1957.

3. Palmer, E. D.: Budd-Chiari syndrome, Ann. Int. Med. 41:261, 1954.

4. Penner, A., and Berheim, A. I.: Acute ischemic necrosis of the kidney, Arch. Path. 30:465, 1940.

PHYSICIANS' ACTIVITIES

A husband and wife team recently opened an office for practice in Lawrence. **Dr. Robert C. Kimbrough, Jr.,** who had been practicing at the Hercules plant at Sunflower, is specializing in internal medicine, and his wife, **Dr. Victoria F. Kimbrough,** is specializing in ophthalmology. He is a graduate of the John Hopkins School of Medicine, and she was graduated from the University of Tennessee. Both had residencies at the University of Michigan School of Medicine.

Dr. Winstan L. Anderson, immediate past president of the Chamber of Commerce in Atchison, was the author of an editorial published in the *Atchison Daily Globe* on January 28. He is now serving as chairman of the Chamber's committee on hotels and motels, and he wrote his editorial on that subject.

Dr. John G. Shellito, Wichita, recently spent a month in Rochester, Minnesota, studying cardiac surgery under Dr. John W. Kirklin.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, served as a member of a panel which selected "America's Ten Outstanding Young Men of the Year" for the United States Junior Chamber of Commerce.

Announcement has been made by **Dr. George Gill** and **Dr. Jack Dysart** of the Trueheart Clinic, Sterling, that **Dr. Theil Bloom,** who will soon complete internship at Wesley Hospital, Wichita, will join the clinic staff. A native of Liberal, Dr. Bloom is a graduate of the University of Kansas School of Medicine.

Dr. Charles P. McCoy has returned to practice in Wichita after having completed a residency in obstetrics and gynecology at St. Louis City Hospital.

Different types of heart disease were described by **Dr. Lyle F. Schmaus,** Iola, when he spoke before the Iola Lions Club recently.

Officers of the Sedgwick County Academy of General Practice, elected for 1958, are: president, **Dr. Jack G. Phipps;** vice-president, **Dr. Robert L. Kasha;** secretary-treasurer, **Dr. Lowell M. Rhodes;** to board of directors for a three-year term, **Dr. Jay K. Wisdom.**

Dr. J. A. Poppen, 80-year-old physician in Burr Oak, has announced plans to retire if another physician can be found to practice in that city.

Dr. E. Grey Dimond, of the University of Kansas Medical Center, was speaker at a Health Education meeting sponsored by the Woman's Auxiliary to the Wyandotte County Medical Society in Kansas City on February 4.

Plans to join **Dr. H. H. Haerle** in practice at the Marysville Clinic were announced last month by **Dr. Lewis R. Laws,** a graduate of the University of Kansas School of Medicine who served his internship at Wesley Hospital in Wichita and recently completed a two-year period of Army service.

Dr. M. M. Tinterow, Wichita, was named alternate director of the American Society of Anesthesiologists for District 15 at the January meeting of the Kansas Society of Anesthesiologists. The district is composed of Kansas and Missouri.

Dr. Russell J. Eilers has joined the staff of the University of Kansas School of Medicine as an associate in pathology. He was graduated from the University of Minnesota School of Medicine in 1953 and took postgraduate training in anatomical and clinical pathology there.

Dr. Victor E. Chesky, Sr., Halstead, has been named honorary chairman of the Halstead Hospital Building Campaign.

Dr. Charles M. Poser, assistant professor of medicine at the University of Kansas Medical Center, recently became a diplomate of the American Board of Psychiatry and Neurology.

A feature story in the *Emporia Gazette* on February 8 paid tribute to **Dr. Charles C. Underwood, Jr.,** chosen as Emporia's "Man of the Week."

Dr. Bernard H. Hall and **Dr. Herbert C. Modlin,** of the Menninger Foundation, Topeka, served as faculty for a seminar on industrial mental health in Chicago recently.

Dr. James G. O'Shea, who has been practicing in Dodge City since 1949, moved to Jetmore last month and is now engaged in practice there.

Physician participants in a health education workshop presented by the Woman's Auxiliary to the Shawnee County Medical Society in Topeka on February 10 were **Dr. David E. Gray** and **Dr. Homer L. Hiebert.**

Dr. D. D. Vermillion, Goodland, was speaker at the annual father-son banquet of the Men's Brotherhood of Emmanuel Lutheran Church in Goodland last month.

Plans to practice in Paola in association with **Dr. Veryl J. Elson** have been announced by **Dr. Robert E. Banks,** a graduate of the University of Kansas School of Medicine who was recently released from service with the U. S. Navy after a tour of duty in Hawaii.

Dr. Curtis A. Nystrom, Cawker City, addressed a rally held in Burr Oak last month to make plans for securing a physician for the Burr Oak community. Dr. Nystrom was induced to begin practice in Cawker City as the result of similar civic effort.

"Neuritis and Neuralgia" was the subject chosen by **Dr. William P. Williamson,** University of Kansas Medical Center, for an address before the Des Moines Medical Library Club recently.

Dr. Harry W. Horn, Wichita, was named senior warden emeritus of St. James Episcopal Church in Wichita recently on his retirement from the vestry. The membership granted the recognition for the first time since Dr. Horn was a founder of the church.

Dr. A. M. Cherner, Hays, was recently made a fellow of the American College of Radiologists.

The Kansas City Star last month took note of the fact that the city of Louisburg is served by two physicians who have each practiced more than half a century there. **Dr. John W. Kelley,** who will be 90 this month, has practiced in Louisburg for 58 years, and **Dr. P. F. Gatley** has been there for 53 years.

Dr. Claude J. Harwood, Glasco, spent several weeks in Kansas City recently to take postgraduate work in anesthesiology.

Dr. Kenneth L. Graham and **Dr. Thomas W. Graham** announce that **Dr. J. Malcolm Graham** is now associated with them in practice in Leavenworth. The new physician will specialize in pediatrics.

Dr. Paul C. Laybourne, Jr., of the University of Kansas Medical Center, spoke on "Prejudice, Its Effect on Children" before a Parent-Teacher Association group in Lawrence last month. He also spoke on "The Management of Reluctance to Attend School" at a meeting of the American Academy of General Practice in Tulsa.

A feature story in the *Hutchinson News-Herald* last month paid tribute to **Dr. Richard A. Stewart** on his 90th birthday. Although he has been in retirement during the past nine years, he retains an interest in the practice of medicine and its advances.

Dr. John W. Fair, acting clinical director at the Parsons State Hospital, was featured speaker at a meeting of the Extension Study Club in Pittsburg last month. More than 200 persons heard Dr. Fair discuss treatment of the mentally retarded and outline the program of the Parsons institution.

"One Success After Another Keys Dr. Wescoe's Career" was the headline given a "profile" of **Dr. W. Clarke Wescoe** in the *Kansas City Kansan* last month. The story told of Dr. Wescoe's work in pharmacology before he became dean of the University of Kansas School of Medicine and then outlined progress at the school under his leadership.

Dr. S. Paul Hornung, speaker at a recent meeting of the Rotary Club in Norton, described the

destructive potential that now exists as the result of scientific developments. Dr. Hornung recently attended a mass casualty management school conducted by the Army in Texas.

Dr. D. D. Fuller, who has been practicing in Marquette, has begun practice in Lindsborg and will return to see patients in Marquette two afternoons each week.

DEATH NOTICES

Thomas Tallman Holt, M.D.

Dr. T. T. Holt, 82, retired Wichita physician, died at St. Francis Hospital, Wichita, on January 18 after an extended illness. A graduate of University Medical College of Kansas City in 1900, Dr. Holt practiced in Geuda Springs until 1922, when he went to New York City for additional training. He then specialized in internal medicine in Wichita and became a diplomate of the American Board of Internal Medicine. He was the first Kansan to become a member of the American College of Physicians, an organization he served as vice-president in 1942. He was also a member of the American Heart Association and the American Chemical Society. Poor health forced his retirement in 1950. Among the survivors is a son, Dr. James H. Holt, Wichita.

William Holmes Dyer, M.D.

A Kansas City physician since 1919, Dr. W. H. Dyer, 71, died on January 21 after suffering a heart attack while driving his car. He had been physician and surgeon for the Santa Fe Railroad for 15 years and had served the Kansas City Police Department in a similar capacity for 29 years. He was graduated from the University of Illinois College of Medicine in 1916 and came to Kansas in 1919, his private practice being interrupted during World War I for service in the Army Medical Corps in France.

William Rolland Palmer, M.D.

Dr. W. R. Palmer, 79, who had practiced in Kansas City almost continuously since his graduation from Kansas City Medical College in 1898, died at his home on January 30. He entered service in World War I as surgeon for the 526th engineer battalion in France. From 1941 through 1945 he served as coroner of Wyandotte County, and during another period he was surgeon for the Kansas City Police Department. He was a member of the Wyandotte County Medical Society.

Hugh Allen Hope, M.D.

A physician who had served as a member of the Kansas State Board of Health and who was prominent in Republican political circles, Dr. Hugh A. Hope, 72, died at Topeka on February 6 while en route from his home at Hunter to Kansas City. A graduate of Kansas Medical College (now Washburn), Topeka, in 1909, Dr. Hope achieved recognition as one of the greatest athletes in the history of the school and had served a term as president of the institution's alumni association. He began medical practice in Victor, moving to Hunter in 1917 as one of the founders of the city. He was an honorary member of the Mitchell County Medical Society.

Porter D. Brown, M.D.

An honorary member of the Saline County Medical Society, Dr. Porter D. Brown, 77, died at Glendale, California, on February 10. After graduation from Kansas Medical College, Topeka, in 1910, Dr. Brown began practice in Alton, moving to Salina about 30 years ago to form a partnership with Dr. L. S. Nelson, Sr. They later organized the Salina Clinic. Dr. Brown, who was a diplomate of the American Board of Obstetrics and Gynecology, retired several years ago and moved to Denver. He had been living in California since last summer.

Harley James Stacey, M.D.

Dr. H. J. Stacey, 87, who had practiced in Leavenworth more than 50 years before his retirement, died January 27 in Oklahoma City, where he had been living for five years. He was graduated from Cornell in 1892 and then entered the University of Pennsylvania School of Medicine, completing that course in 1896. He was the fourth physician to join the staff of the Cushing Hospital in Leavenworth. At the time of his death Dr. Stacey was an honorary member of the Leavenworth County Medical Society.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Those who are trying to follow the course of medical legislation find an unusual situation developing in this session of Congress. All of Washington is being subjected to forces, some completely new, that often work at cross-purposes to each other. The result could be a moratorium on health legislation—or again it could be a flood of new laws.

At the start of the session, a new-born interest in science completely dominated the scene— by a frantic spending of billions of dollars we would overtake Russia. That was the theme in Washington, and it persisted despite a few quiet voices that asked whether Russia really had far outdistanced the U. S. or was merely exploiting a slight advantage.

Even before the American satellite started on its orbit, some of the panic had subsided, and most of the legislators had decided that advent of the space age had not removed all of the old problems and opportunities in legislation and politics. The familiar issues were still there, medical panaceas included.

The shock of Russian achievements will, at any rate, produce legislation designed to shore up our educational system. This seems to be generally accepted. For the medical profession, two provisions are of major interest. Scholarships would be for four years—possibly six—offering some assistance to premedical students and in some cases to those in the first year of medical school. Also, fellowships would be available for medical and other graduates if they wanted to teach or go into research.

The administration's idea was a program that would cost a billion dollars; several leading Democrats joined in a bill proposing three billion dollars as a stimulant to mathematics and science.

But there are other factors to be reckoned with. For the first time a president set down in black and white in his budget just how he proposed to withdraw the federal government from some activities, or limit its participation, and turn the programs back to the states. Mr. Eisenhower wants to slow down on the Hill-Burton hospital construction program and change its emphasis, he wants to mesh in some veterans' benefits with social security payments, he would have the states do more and the U. S. less in public assistance (where medical payments are a growing factor), and he hopes to get Congress to drop the $50 million a year program of grants to help build water treatment plants.

Whether Congress will follow the President's lead in the back-to-the-states movement is another question. At least he has said specifically what he thinks should be done, and when.

There was no expectation that the Russian scare would dilute politics this election year—and it hasn't. If anything, the partisans are struggling harder than ever to make records that will reflect glory on them next November. Some, of course, would be pressing for their projects regardless of the election.

So this is the prospect, in brief:

The Defense Department and science will get the major attention and the major money, but some may spill over into medicine.

There is some interest in a tight domestic budget and returning certain activities to the states, but old fashioned politics combined with a fear of a continuing recession may again open up the federal purse.

Medical legislation, always a popular subject, may get more and more attention as the session rolls on. If so, the Forand bill among others would come immediately to the fore.

Notes

Several developments in the legislative field on Jenkins-Keogh bills came early in the session. The American Thrift Assembly, representing some 10 million self-employed, urged favorable House Ways and Means action, and the American Medical Association pointed out that the proposal for tax deferment of money paid into retirement plans could help solve the problem of maldistribution of physicians.

In the Senate, a majority of the Small Business Committee introduced a tax relief bill with a J-K provision. The section would allow anyone not now benefiting from a qualified pension plan to set aside 10 per cent of annual income ($1,000 maximum). The bill went to the Senate Finance Committee.

A limited number of medical scientists from this country and Russia will give lectures in each other's countries this year in an exchange program worked out by the State Department and the Soviet government. Also planned are exchanges of medical journals between medical libraries and of medical films. All these are part of a broad scientific; cultural, and education program between the two nations. Details haven't been worked out.

Medical Education Week in April

Each member of the Kansas Medical Society has an opportunity this month to both honor and aid his medical school by helping bring the third annual observance of Medical Education Week to the attention of his patients and the public.

During the week of April 20-26, the medical profession will join forces with the Woman's Auxiliary and the medical schools throughout the country in presenting programs emphasizing the progress, problems, and challenges of medical education. The world leadership of American medical schools, their expanding enrollments, research triumphs, and community services are little known by the public at large, and Medical Education Week is designed to create greater public appreciation and support for their continuing achievements. At the same time, it will stress the problems which the foreseeable future holds—increased competition for the qualified school candidate, greater facilities for teaching the growing complexities of medicine, and the need of an expanding and aging population for more doctors. And, not least of all, is the immense cost of medical education which already is a $200 million annual undertaking.

The six specific aims of Medical Education Week, which our society, in cooperation with our Woman's Auxiliary and the University of Kansas School of Medicine will stress through local radio and television programs, newspaper features, and presentations to community organizations, are to:

1. Portray the key role that medical education plays in the promotion and maintenance of the nation's health and security, and make the public aware that the nation's 83 medical schools are the foundation of our entire health and medical structure;

2. Explain how medical schools are striving to meet the demand for larger numbers of physicians and, at the same time, characterize American medical education;

3. Call attention to the steady progress in the medical sciences, showing what this means in terms of longer life, better health, and greater freedom from disease and disability;

4. Point out the wide range of activities—teaching, research, service, and leadership—carried on by the modern medical school in addition to its job of training new doctors;

5. Make clear the extent and nature of the new challenges to the profession, some growing out of our constantly expanding fund of medical knowledge and some resulting from the mounting complexity of our civilization, and

6. Point out some of the steps being taken constantly to push back the horizons of the medical sciences and to realize the full potential of the nation's health resources.

These objectives will be further emphasized in a coast-to-coast promotional effort now being planned by the national sponsors of Medical Education Week —the Association of American Medical Colleges, the A.M.A. and its Woman's Auxiliary, the Student A.M.A., the National Fund for Medical Education, and the American Medical Education Foundation. Programs and salutes, supplementing our local observance, will be carried on network radio and television, in national publications and syndicated newspaper features, and through civic and industrial organizations.

President Eisenhower, in his personal endorsement, has already invited the American people to set aside this week to consider the work of our medical schools, but its ultimate success will depend most directly on how well and how actively we initiate and conduct this annual community salute to our medical schools . . . Medical Education Week, April 20-26!

Basic Science Examination

The Kansas Board of Basic Science Examiners will give examinations in the subjects of anatomy, bacteriology, chemistry, pathology, and physiology on June 3 and 4, 1958, in the auditorium of Wahl Hall, University of Kansas Medical Center, Kansas City. Satisfactorily completed applications for examination must be submitted at least 30 days prior to the examination. Application blanks and other information may be obtained from R. E. Stowell, M.D., secretary of the Kansas Board of Basic Science Examiners, Kansas City 12, Kansas.

Legal Conference in May

Legal problems currently facing individual physicians and organized medicine will be the primary discussion topics at the second meeting of state and county medical society executive secretaries and attorneys, May 9-10, at the Drake Hotel, Chicago. Before the final agenda can be set up, the A.M.A. Law Department hopes that medical societies will send in their suggestions on specific legal subjects that would be of the most interest to them. The first such meeting—also sponsored by the Law Department—was held in April, 1956.

In 1956, there were 1,280 persons killed in U. S. train-car crashes.

A total of 40,000 Americans were killed in 1956 traffic accidents.

BOOK REVIEWS

The Relation of Psychiatry to Pharmacology. By Abraham Wikler. Published by American Society for Pharmacology and Experimental Therapeutics by Williams and Wilkins Company, Baltimore. 322 pages. Price $4.00.

Interest in modern psychotherapeutic drugs has not only pervaded the medical profession but has spilled over into the popular press and even into radio and television. At least one book of modern humor, the title of which includes the name of a well-known tranquilizer, is selling well—no doubt partly because of its name. Of importance to the scientific world is the fact that the new psychotropic drugs are at least in some degree responsible for a renaissance of interest in the fields of neurophysiology, psychophysiology, and psychopharmacology—three intimately related areas which are in need of more work.

Dr. Wikler's book is a review of the literature dealing with these fields and relating them to experimental psychology. It was originally conceived as an article for *Pharmacological Reviews,* but the size of the manuscript made it unsuitable for publication in that form, so it appears as a monograph under the imprimatur of the American Society for Pharmacology and Experimental Therapeutics. It seems to this reviewer that the title should actually be "The Relation of Pharmacology to Psychiatry" because the text is concerned with relating the innumerable fragments of knowledge of the basic sciences to the causes and treatment of mental illness. The book is divided into four main sections, the first of which deals with the effect of drugs on human behaviour. Subsequent sections are concerned with theories and mechanisms of drugs actions, the neurophysiological aspects of drugs, and the psychological implications.

The organization as reflected by the table of contents is satisfactory, but the organization within some of the sections is not so good. This is not to be wondered at, considering the tremendous mass of material dealt with, but it is all the more important to the reader. The paucity of sub-headings and paragraph headings does not ease the task of one searching for specific items, and the excessively long paragraphs not only contribute to the reader's difficulty but to his irritation. There is simply no excuse for routinely writing paragraphs of one and two pages, and a paragraph three and one-half pages long should never have been permitted by the editors.

The author reports almost everything that has ever been written on the subject, and one wonders whether he might have selected some of his material more critically. It would seem to be the obligation of an author of a review article to point out disproved work or to select his citations with extreme care.

The book is a gold mine of information, but the reader has to mine it for himself, refine the ore, and smelt it. It is obviously not a book for the average physician or even for the average psychiatrist, but it should be an extremely useful source book for research men in psychiatry, physiology, and pharmacology. There are 889 references. The index is probably adequate, but not as complete as one could wish. The printing is excellent and on a soft finish paper which makes reading easy. The binding is excellent for a paperback volume.—*J.D.R.*

One Surgeon's Practice. By Frederick Christopher, M.D. Published by W. B. Saunders Company, Philadelphia. 151 pages. Price $4.00.

In this short book Dr. Christopher in no way attempts to make it a scientific treatise. He rather looks back over his years of practice and preparation and attempts to describe this preparation, training, development of practice, etc.

It is interesting reading for a practicing doctor although it does not in any way add to his knowledge of the practice of medicine. I would rather recommend the book to the medical student or the young man or woman thinking of or planning to enter the medical field.—*S.L.V.*

A Textbook of Clinical Neurology. By Israel S. Wechsler, M.D. Published by W. B. Saunders Company, Philadelphia. 782 pages, 179 figures. Price $11.

This eighth edition, like all its predecessors, pays tribute to the brisk and readable style in which this renowned American neurologist presents information on his specialty. Again, also, the worth-while chapters describing psychological testing and psychiatric syndromes are added to the conventional sections on neurologic examination, clinical neurologic syndromes, etc. A worth-while further addition is a chapter dedicated to the history of neurology and the contributions of the now countless contributors to the field.

This eighth edition continues the tradition of this well-known text as a standard in the library of all those interested in the field.—*J.A.S.*

Digitalis. Compiled and edited by E. Grey Dimond, M.D. Published by Charles C Thomas, Publisher, Springfield, Illinois. 255 pages. Price $7.00.

This monograph on digitalis includes material presented in a two-day postgraduate course given in February 1956, plus additional papers contributed by

Dr. Ralph H. Major, Dr. Meyer Friedman, and Dr. George Okita. The contributors who participated as faculty for the postgraduate session were Robert Batterman, Richard Bing, E. Grey Dimond, Santiago Grisolia, Bernard Lown, Aldo Luisada, and William Sodeman, all of whom are working in this field in the United States. The final chapter is a panel discussion by the same group with Dr. Dimond as moderator.

The first chapter of this volume is a reprint from Major's *Classic Descriptions of Disease,* a splendid introduction!

The chapters discussing the pharmacology of digitalis and the more active glycosides with reference to their physiological actions are especially interesting and instructive to the experienced physician and the recent graduate.

Two chapters, *Observation on the Clinical Use of Digitalis* by Robert C. Batterman and *Problems of Bedside Management of Digitalis* are of unusual interest because of their practical value. The discussion of the relationship of potassium and digitalis is pertinent and informative. The chapter on auricular arrhythmias should be read by every physician who uses digitalis. The discussion on the possible wider range between therapeutic effect and toxicity with one of the cardiac glycosides is of note, especially since all the participants of the panel did not concur.

The discussion of the use of acetyl strophanthidin in ascertaining the presence or absence of digitalis intoxication is valuable for one who has time to observe the procedure carefully. The caution against the indiscriminate use of intravenous digitalis preparations is timely.

Dr. Lown's final comments are good because he emphasizes that an initial admission electrocardiogram will not always tell whether a patient has been taking digitalis. His quote from Calvin Kay "The fallacies of clinical judgment and of patients' memory are better avoided by means of a telephone call than by means of an electrocardiogram."

This volume would be a welcome addition to any physician's library.—*C.C.U.*

ANNOUNCEMENTS

Courses at University of Kansas Medical Center soon: Otolaryngology, April 9-11; Ophthalmology, April 7-9; Anesthesiology, April 14-16.

Physicians and Surgeons Book Company, Henry and Pacific Streets, Brooklyn, New York, will purchase medical books. Send list of books giving author, title, and date of publication. Company will respond with offer for books they desire to buy.

Daniel B. Kirby fellowship for research in ophthalmology offered by Department of Ophthalmology, New York University-Bellevue Medical Center, 550 First Avenue, New York 16, New York. Appointment for one year at stipend of $7,000, subject to renewal for two additional years. Applications accepted until May 1, 1958.

The General Practitioner Education Project, jointly sponsored by the American Psychiatric Association and the American Academy of General Practice, offers services of a speakers' bureau. Names of psychiatrists willing to serve as guest lecturers while taking vacation trips available to medical societies, hospitals, etc. Write G. P. Project, American Psychiatric Association, 1785 Massachusetts Avenue, N. W., Washington, D. C.

Postgraduate course on clinical physical medicine, University of Colorado Medical Center, May 5-7, 1958. Write the Center, 4200 East Ninth Avenue, Denver 20, Colorado.

Fellowships offered by National Foundation for Infantile Paralysis, 301 East 42nd Street, New York 17, New York.

World Congress of Gastroenterology, Sheraton-Park Hotel, Washington, D. C., May 25-31. Host organization, American Gastroenterological Association, c/o H. M. Pollard, M.D., University Hospital, Ann Arbor, Michigan.

Congress and graduate instructional course in allergy, Shelburne Hotel, Atlantic City, New Jersey, April 20-25, under auspices of American College of Allergists, 2049 Broadway, Boulder, Colorado (c/o John D. Gillaspie, M.D., treasurer).

Sixth annual interim scientific meeting of Phi Lambda Kappa, national medical fraternity, Deauville Hotel, Miami Beach, Florida, April 13-20. Write Dr. Samuel L. Lemel, 1030 Euclid Avenue, Cleveland 15, Ohio.

Trip to Hawaii planned by Illinois State Medical Society at close of annual meeting of American Medical Association in San Francisco in June. Party will leave by air night of June 26, return by air on July 5 or by *S.S. Lurline* on July 10. Write Mr. W. M. Moloney, Professional Building, "Old Orchard," Skokie, Illinois.

Venereal Disease Postgraduate Conference for physicians sponsored by University of Texas, Baylor, Texas State Department of Health, Texas Medical Association, and USPHS, at M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston, April 23-25. Twenty hours AAGP credit.

Award of $500 offered by editors of *Modern Medical Monographs* for best unpublished manuscript for a short book on a clinical subject in field of internal medicine. Deadline October 1, 1958. Write Richard H. Orr, M.D., 37 East 67th Street, New York 21, New York.

Council of World Medical Association, Paris, France, May 3-10; General Assembly, Parliament House, Copenhagen, Denmark, August 15-20. Write World Medical Association, 10 Columbus Circle, New York 19, New York.

"Principles and Practice of Internal Medicine" is subject of course to be presented at State University of Iowa College of Medicine, Iowa City, June 2-6 under auspices of American College of Physicians, 4200 Pine Street, Philadelphia 4, Pennsylvania.

COUNTY SOCIETIES

Dr. L. L. Cooper of Fort Scott was named president of the Bourbon County Medical Society at a meeting held last month, succeeding Dr. J. R. Prichard who had held the office for 15 years. Dr. Pratt Irby became vice-president and Dr. James J. Basham, secretary.

"The Immediate Etiological Diagnosis in the Convulsing Patient" was the subject chosen by Dr. Larry Calkins, of the University of Kansas Medical Center, who addressed the Shawnee County Medical Society in Topeka on February 10.

A meeting of the Wyandotte County Society was held at the Town House Hotel on February 18. Speaker was Mr. Mac F. Cahal, executive secretary of the American Academy of General Practice, who discussed medical public relations.

Members of the Leavenworth County Society entertained their wives and members of the Leavenworth School Board at a dinner meeting at the Hotel Cody on February 8. Mr. C. A. Christ, principal of the junior high school, spoke on "Problems Facing the School of Today."

Dr. Peter A. Rosi, associate professor of surgery at Northwestern University Medical School, Chicago, discussed "Current Concepts of Surgery of the Colon" before the Sedgwick County Society at a meeting in Wichita on February 4.

Officers of the Iroquois Medical Society, elected at a meeting held in January in Greensburg, are: Dr. Lyle G. Glenn of Protection, president; Dr. Ronald McCoy, Coldwater, vice-president, and Dr. William W. Orrison, Plains, secretary.

Two New A.M.A. Exhibits

Reducing and accidental poisoning of children are the themes of two new exhibits the American Medical Association is offering to medical societies early in 1958. "You Can Reduce" stresses the importance of using will power in the selection of foods. The exhibit illustrates the basic foods that should be eaten every day, those to "fill up" on and those to "cut down" on. Three dimensional models depict the calorie content of certain basic foods. "Poisoning of Children in the Home" pinpoints eight leading offenders, such as aspirin, kerosene, old medicines, and household chemicals. A display of products on a revolving tree-like arrangement also is included in this portable exhibit. Medical society bookings may be arranged through the Bureau of Exhibits.

CLASSIFIED ADVERTISEMENTS

FOR SALE—X-ray machine, General Electric, 100 MA with fluoroscope. One tube. In central Kansas. Write the JOURNAL 2-58.

POSITION WANTED—locum tenens or position as assistant to established man. Available from early January through June. Excellent general training, married, have family. Prefer location in or around Kansas City area. Recent graduate. Write the JOURNAL 1-58.

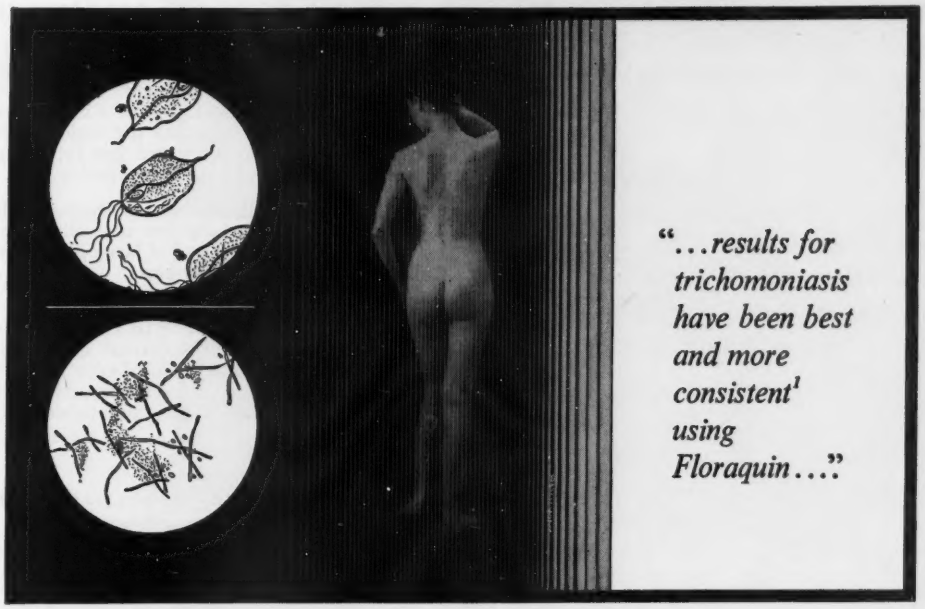
Progressive, growing group of six physicians would like to contact an obstetrician, pediatrician, internist, and general practitioner who have completed military obligations. Very prosperous central Nebraska town under 20,000 offers unlimited future possibilities. Excellent salary first year, attractive partnership thereafter. Write the Journal 3-58.

FOR SALE OR LEASE—7-room air-conditioned office recently built. Lucrative practice in rich farming and industrial area in Nebraska. New, open-staff hospital in town. Wish to complete board training. Leaves town with no surgeon. Territory and population warrant two doctors. Write the JOURNAL 5-58.

LABORATORY X-RAY TECHNICIAN wanted in Belleville. Work in hospital and in private office. Write the JOURNAL 6-58.

WANTED LOCUM TENENS—one year beginning January 1, 1958. Physician interested in industrial medicine and limited general practice. Salary open. Write the JOURNAL 7-58.

EFFECTIVE, DEPENDABLE THERAPY FOR VAGINITIS



Floraquin® eliminates trichomonal and mycotic infection; restores normal vaginal acidity

Leukorrhea is by far the most frequent symptom of vaginitis; trichomonads and monilia are the most common causes. Many authors have reported[2] trichomonal protozoa in the vagina of 25 per cent of obstetric and gynecologic patients. Increased use of broad spectrum antibiotics has resulted in a sharp rise in the incidence of monilial infections.

Floraquin effectively eradicates both trichomonal and monilial vaginal infections through the action of its Diodoquin® content. Floraquin also furnishes boric acid and sugar to restore the normal vaginal acidity which inhibits pathogens and favors the growth of protective Döderlein bacilli.

Pitt[1] recommends vaginal insufflation of Floraquin powder daily for three to five days, followed by acid douches and the daily insertion of Floraquin vaginal tablets throughout one or two menstrual cycles. G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

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1. Pitt, M. B.: Leukorrhea. Causes and Management, J. M. A. Alabama *25*:182 (Feb.) 1956.
2. Parker, R. T.; Jones, C. P., and Thomas, W. L.: Pruritus Vulvae, North Carolina M. J. *16*:570 (Dec.) 1955.

SEARLE

Hospital practice of infant feeding

Standard formulas for PREMATURES

Breast milk is satisfactory for the feeding of prematures in spite of the low protein and mineral and high fat content. But eventual formula feeding should provide a high protein and carbohydrate to satisfy the rapid-growing needs of the premature and low fat content because of limited digestive capacity.

Feedings of small prematures are most effectively administered by the indwelling polythene nasal catheter and of large prematures, by bottle with small nipples.

The first six feedings should be a sterile 5% solution of Karo Syrup at 2 to 3 hour intervals; for subsequent feedings, breast milk or formula should be added in gradually increasing amounts according to tolerance and requirements, as indicated in the table below.

Initial feeding schedules for premature infants

(Feedings Started After 36 Hours and Continued at 2 to 3 Hour Intervals)

FEEDINGS	COMPOSITION	QUANTITY
First Six	5% Karo	2-5 ml.
7th and 8th	2 parts 5% Karo 1 part breast milk or formula	6-10 ml.
9th and 10th	1 part 5% Karo 1 part breast milk or formula	8-15 ml.
11th and 12th	1 part 5% Karo 2 parts breast milk or formula	10-18 ml.
Subsequently	Breast or formula feeding	12-20 ml.

ADVANTAGES OF KARO® IN INFANT FEEDING

Composition: Karo is a superior maltose-dextrin mixture because the dextrins are non-fermentable and the maltose is rapidly transformed into dextrose which requires no further digestion.

Concentration: Volume for volume Karo furnishes twice as many calories as similar milk modifiers in powdered form.

Purity: Karo is processed at sterilizing temperatures, sealed for complete hygienic protection and devoid of pathogenic organisms.

Low Cost: Karo costs 1/5th as much as expensive milk modifiers and is available at all food stores.

Medical Division
CORN PRODUCTS REFINING COMPANY
17 Battery Place, New York 4, N.Y.

MY DAD— HE HURT HIS BACK REAL BAD

"It happened at work while he was putting oil in something"

"He told Mom his shoulder felt like it was on fire"

"He couldn't swing a bat without hurting"

"But Doctor gave him some nice pills--and the pain went away fast"

"Dad said we'd play ball again tomorrow when he comes home"

AND THE PAIN WENT AWAY FAST

FOR PAIN

Percodan®

(Salts of Dihydrohydroxycodeinone and Homatropine, plus APC) **TABLETS**

ACTS FASTER...
usually within 5-15 minutes

LASTS LONGER...
usually for 6 hours or more

MORE THOROUGH RELIEF...
permits uninterrupted sleep through the night

RARELY CONSTIPATES...
excellent for chronic or bedridden patients

and now... **NEW**

Percodan®-Demi

VERSATILE

New "demi" strength permits dosage flexibility to meet each patient's specific needs. PERCODAN-DEMI provides the PERCODAN formula with one-half the amount of salts of dihydrohydroxycodeinone and homatropine.

AVERAGE ADULT DOSE: 1 tablet every 6 hours. May be habit forming. Available through all pharmacies.

Each PERCODAN Tablet contains 4.50 mg. dihydrohydroxycodeinone hydrochloride, 0.38 mg. dihydrohydroxycodeinone terephthalate, 0.38 mg. homatropine terephthalate, 224 mg. acetylsalicylic acid, 160 mg. phenacetin, and 32 mg. caffeine.

Literature? Write

Endo® **ENDO LABORATORIES**
Richmond Hill 18, New York

U.S. Pat. 2,628,185

little How to win friends ...

The Best Tasting Aspirin you can prescribe.

The Flavor Remains Stable down to the last tablet.

25¢ Bottle of 48 tablets (1¼ grs. each).

We will be pleased to send samples on request.

THE BAYER COMPANY DIVISION of Sterling Drug Inc. 1450 Broadway, New York 18, N. Y.

there is one tranquilizer clearly indicated in peptic ulcer...

*Tests in a series of 25 patients show that there is "a definite and distinct lowering [of both volume of secretions and of free hydrochloric acid] in the majority of patients. . . . No patients had shown any increase in gastric secretions following administration of the drug."[1]

Now you have 4 advantages when you calm ulcer patients with ATARAX:

1. ATARAX suppresses gastric secretions; others commonly increase acidity.
2. ATARAX is "the safest of the mild tranquilizers."[2] (No parkinsonian effect or blood dyscrasias ever reported.)
3. It is effective in 9 of every 10 tense and anxious patients.
4. Five dosage forms give you maximum flexibility.

supplied: 10, 25 and 100 mg. tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

references: 1. Strub, I. H.: Personal communication. 2. Ayd, F. J., Jr.: presented at Ohio Assembly of General Practice, 7th Annual Scientific Assembly, Columbus, September 18-19, 1957.

Roerig Rx
New York 17, New York
Division, Chas. Pfizer & Co., Inc.

NOW...A NEW TREATMENT

'CARDILATE'*

for THE PROPHYLAXIS OF ANGINA PECTORIS

'Cardilate' tablets shaped for easy retention in the buccal pouch

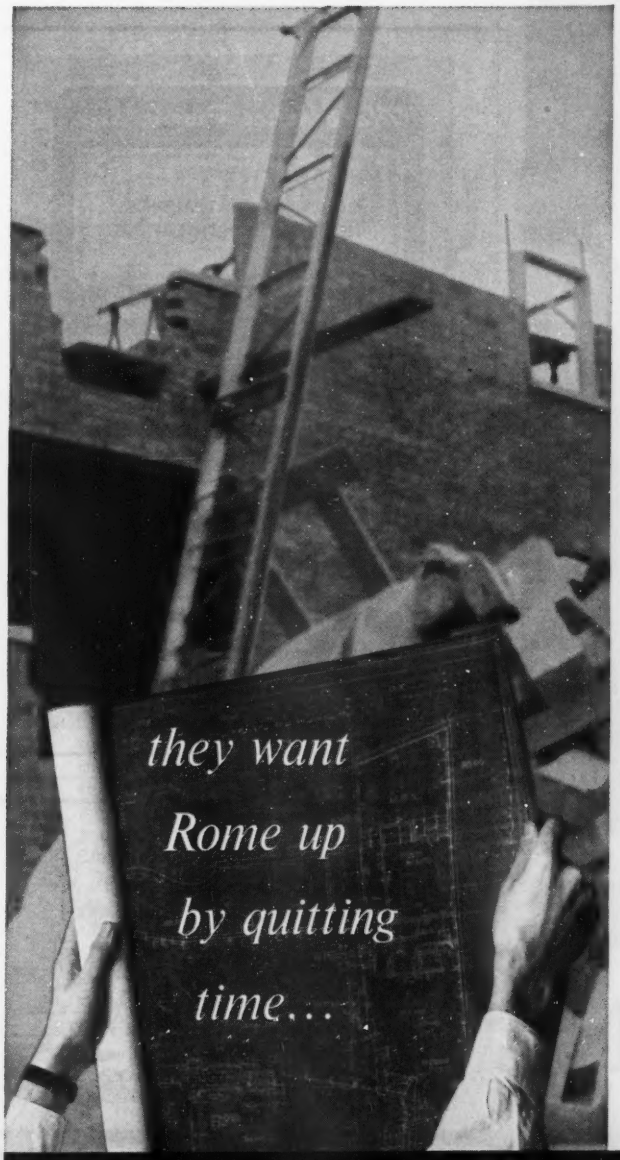
"... the degree of increase in exercise tolerance which sublingual erythrol tetranitrate permits, approximates that of nitroglycerin, amyl nitrite and octyl nitrite more closely than does any other of the approximately 100 preparations tested to date in this laboratory."

"Furthermore, the duration of this beneficial action is prolonged sufficiently to make this method of treatment of practical clinical value."

Riseman, J. E. F., Altman, G. E., and Koretsky, S.: Nitroglycerin and Other Nitrites in the Treatment of Angina Pectoris, Circulation (Jan.) 1958.

*'Cardilate' brand Erythrol Tetranitrate SUBLINGUAL TABLETS, 15 mg. scored

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York



To cut daytime lethargy

(and keep rauwolfia potency)

in treatment of hypertension:

Mounting clinical evidence confirms the view that HARMONYL produces much less lethargy while reducing blood pressure effectively. In the most recent study[1], HARMONYL was evaluated in comparison with reserpine and other rauwolfia alkaloids. HARMONYL *was the only alkaloid which produced a hypotensive response closely matching that of reserpine, coupled with a greatly reduced rate of lethargy.* Only one HARMONYL patient in 20 showed lethargy, while an average of 11 out of 20 showed lethargy with reserpine, and 10 out of 20 with the alseroxylon fraction. Abbott

ED2077

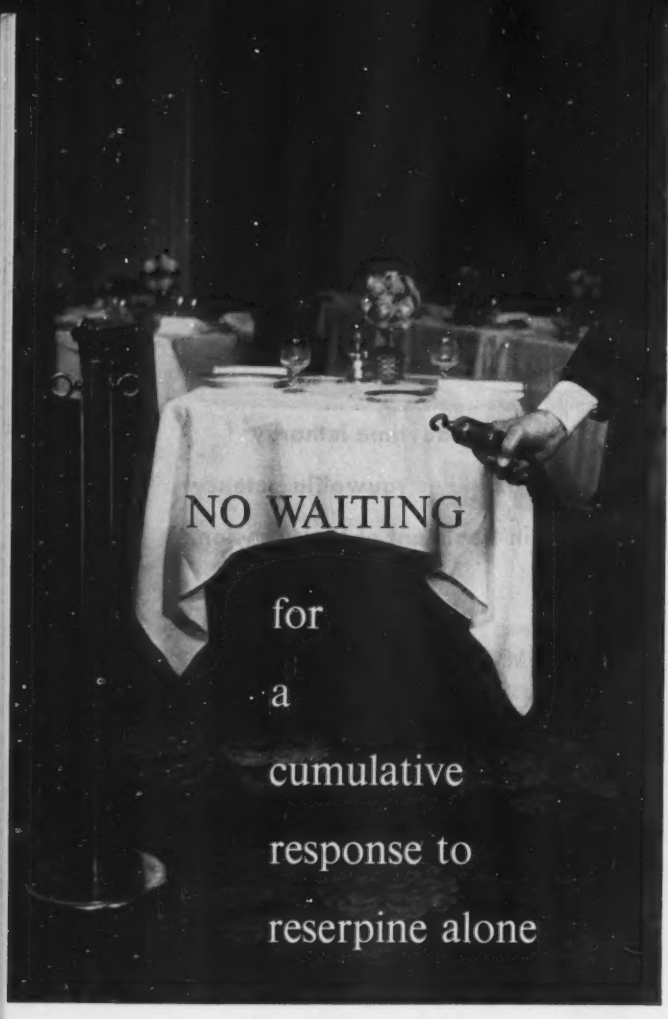
for your hypertensives who must stay on the job

Harmonyl

(DESERPIDINE, ABBOTT)

while the drug works effectively . . . so does the patient

1. Comparative Effects of Various Rauwolfia Alkaloids in Hypertension. *Diseases of the Chest*, in press.



in anxiety and hypertension
NEW *fast-acting*

filmtab®

Harmonyl-N*

(Harmonyl* and Nembutal®)

Calmer days, more restful nights starting *first* day of treatment, through synergistic action of HARMONYL (Deserpidine, Abbott) and NEMBUTAL (Pentobarbital, Abbott). Lower therapeutic doses, lower incidence of side effects. Each HARMONYL-N Filmtab contains 30 mg. NEMBUTAL Calcium and 0.25 mg. HARMONYL. Each HARMONYL-N *Half-Strength* Filmtab combines 15 mg. NEMBUTAL Calcium and 0.1 mg. HARMONYL. Abbott

®Filmtab—Film-sealed tablets, Abbott; pat. applied for

901000 *Trademark

Malpractice Prophylaxis

COVERAGE THAT SUFFICES,
NOT THAT WHICH ENTICES

Specialized Service
makes our doctor safer

THE
MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA

Professional Protection Exclusively
since 1899

KANSAS CITY Office:
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Protection Against Loss of Income From Accident and Sickness as Well as Hospital Expense Benefits for You and All Your Eligible Dependents.

ALL PREMIUMS COME FROM PHYSICIANS SURGEONS DENTISTS ALL BENEFITS GO TO

PHYSICIANS CASUALTY & HEALTH ASSOCIATIONS
OMAHA 31, NEBRASKA
Since 1902

STOP respiratory congestion orally

relief in minutes..lasts for hours

In the common cold, nasal allergies, sinusitis, and postnasal drip, one timed-release Triaminic tablet brings welcome relief of symptoms *in minutes*. Running noses stop, clogged noses open—and *stay* open for 6 to 8 hours. The patient can breathe again.

With *topical* decongestants, "unfortunately, the period of decongestion is often followed by a phase of secondary reaction during which the congestion may be equal to, if not greater than, the original condition. . . ."* The patient then must reapply the medication and the vicious cycle is repeated, resulting in local overtreatment, pathological changes in nasal mucosa, and frequently "nose drop addiction."

Triaminic does not cause secondary congestion, eliminates local overtreatment and consequent nasal pathology.

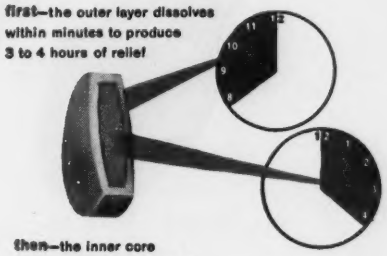
*Morrison, L. F.: Arch. Otolaryng. 59:48-53 (Jan.) 1954.

Each double-dose "timed-release" TRIAMINIC *Tablet contains:*

Phenylpropanolamine hydrochloride	50 mg.
Pyrilamine maleate	25 mg.
Pheniramine maleate	25 mg.

Dosage: 1 tablet in the morning, afternoon, and in the evening if needed.

Each double-dose "timed-release" tablet keeps nasal passages clear for 6 to 8 hours—provides "around-the-clock" freedom from congestion on just three tablets a day


first—the outer layer dissolves within minutes to produce 3 to 4 hours of relief
then—the inner core disintegrates to give 3 to 4 more hours of relief

Also available: Triaminic Syrup, for children and those adults who prefer a liquid medication.

Triaminic®

"timed-release" tablets

STOP *running noses... and open stuffed noses orally*

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska • Peterborough, Canada

there's pain and inflammation here... it could be mild or severe, acute or chronic, primary or secondary fibrositis—or even early rheumatoid arthritis

more potent and comprehensive treatment than salicylate alone

. . . assured anti-inflammatory effect of low-dosage corticosteroid[1]

. . . additive antirheumatic action of corticosteroid plus salicylate[2-5] brings rapid pain relief; aids restoration of function.

. . . wide range of application including the entire fibrositis syndrome as well as early or mild rheumatoid arthritis

more manageable corticosteroid dosage

. . . much less likelihood of treatment-interrupting side effects[1-6]

. . . simple, flexible dosage schedule

in any case
it calls for

Sigmagen®

corticoid-salicylate compound

tablets

Composition

METICORTEN® (prednisone)	0.75 mg.
Acetylsalicylic acid	325 mg.
Aluminum hydroxide	75 mg.
Ascorbic acid	20 mg.

Packaging: SIGMAGEN Tablets, bottles of 100 and 1000.

References: 1. Spies, T. D., et al.: J.A.M.A. 159:645, 1955. 2. Spies, T. D., et al.: Postgrad. Med. 17:1, 1955. 3. Gelli, G., and Della Santa, L.: Minerva Pediat. 7:1456, 1955. 4. Guerra, F.: Fed. Proc. 12:326, 1953. 5. Busse, E. A.: Clin. Med. 2:1105, 1955. 6. Sticker, R. B.: Panel Discussion, Ohio State M. J. 52:1037, 1956.

Acute conditions: Two or three tablets four times daily. After desired response is obtained, gradually reduce daily dosage and then discontinue.

Subacute or chronic conditions: Initially as above. When satisfactory control is obtained, gradually reduce the daily dosage to minimum effective maintenance level. For best results administer after meals and at bedtime.

Precautions: Because SIGMAGEN contains prednisone, the same precautions and contraindications observed with this steroid apply also to the use of SIGMAGEN.

SCHERING CORPORATION • BLOOMFIELD, N. J.

60-J-648

Schering

IN ALL DIARRHEAS . . . REGARDLESS OF ETIOLOGY

comprehensive control with **CREMOMYCIN**®

SULFASUXIDINE® PECTIN-KAOLIN-NEOMYCIN SUSPENSION

SOOTHING ACTION . . . Kaolin and pectin coat and soothe the inflamed mucosa, adsorb toxins and help reduce intestinal hypermotility.

BROAD THERAPY . . . The combined antibacterial effectiveness of neomycin and Sulfasuxidine is concentrated in the bowel since the absorption of both agents is negligible.

LOCAL IRRITATION IS REDUCED and control is instituted against spread of infective organisms and loss of body fluid.

PALATABLE creamy pink, fruit-flavored CREMOMYCIN is pleasant tasting, readily accepted by patients of all ages.

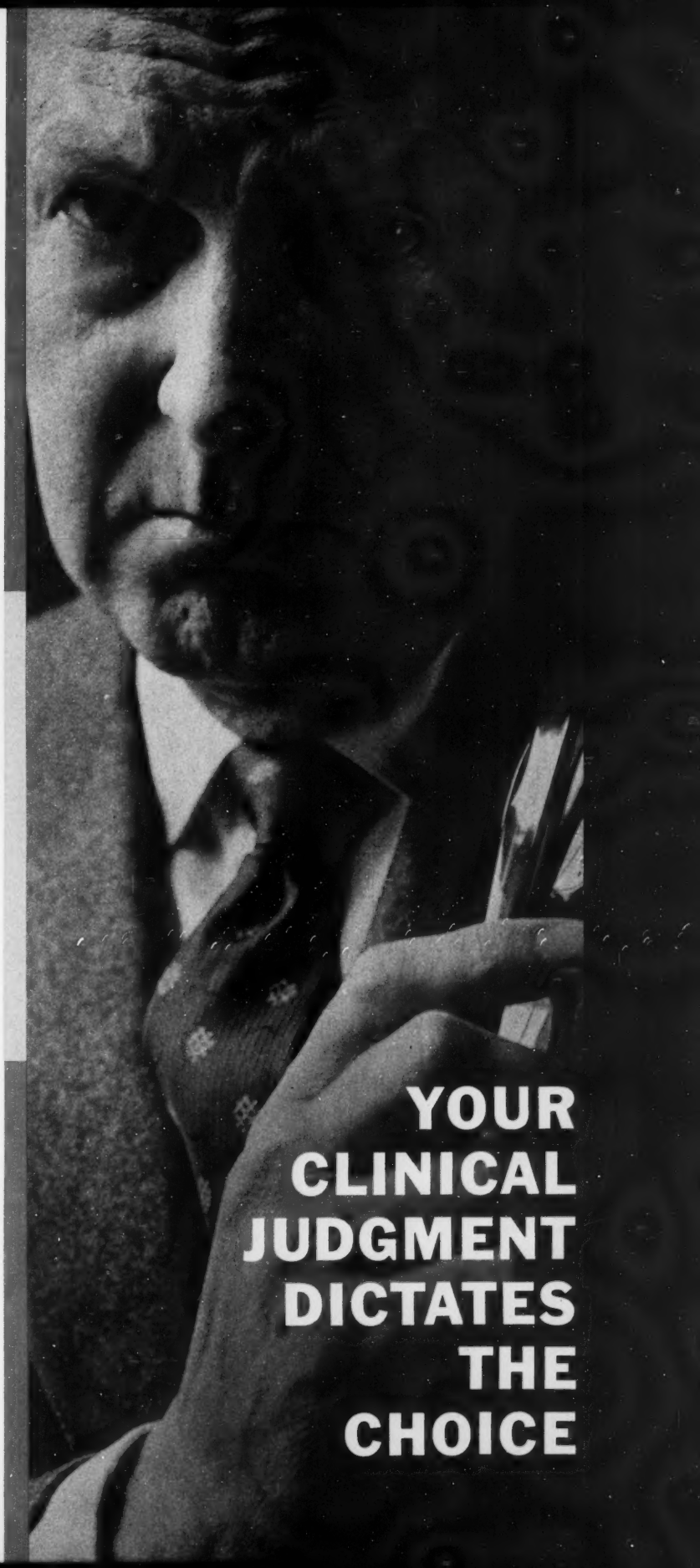
* Sulfasuxidine is a trade-mark of Merck & Co., Inc.

MSD **MERCK SHARP & DOHME**
DIVISION OF MERCK & CO., INC., PHILADELPHIA 1, PA.

in common mixed infections
...tetracycline phosphate alone

in potentially serious infections
...tetracycline phosphate plus novobiocin

for the 7 monilia-susceptible types
...tetracycline phosphate plus nystatin



YOUR CLINICAL JUDGMENT DICTATES THE CHOICE

in common mixed infections
...tetracycline phosphate alone

PANMYCIN Phosphate

for children:

PANMYCIN Syrup

in potentially serious infections
...tetracycline phosphate plus novobiocin

PANALBA*

for children:

PANALBA Granules

TRADEMA

for the 7 monilia-susceptible types
...tetracycline phosphate plus nystatin

COMYCIN*

Upjohn

The Upjohn Company, Kalamazoo, Michigan

*TRADEMARK, REG. U. S. PAT. OFF.

CIN*
te

CIN KM
TRADEMARK

BROAD-SPECTRUM TETRACYCLINE IN ITS MOST EFFICIENT FORM

Produces more tetracycline in the blood with no more in the dose. No calcium to depress blood levels.[1] Basic broad-spectrum therapy in bronchitis, pharyngitis, otitis media, tonsillitis, and other common respiratory infections.

1. Welch, H.; Wright, W. W.; and Staffa, A. W.: Antibiotic Med. & Clin. Therapy 4:620, 1957.

BA*

BA KM
TRADEMARK
S

THE BREADTH OF PANMYCIN PHOSPHATE PLUS THE ANTIMICROCOCCAL DEPTH OF ALBAMYCIN†

Offers maximum antimicrobial action at the earliest possible moment. The antibiotic preparation of first resort in pneumonia of unknown etiology, carbuncles, multiple furunculosis, cellulitis, and infections resistant to previous therapy.

†TRADEMARK, REG. U. S. PAT. OFF. — THE UPJOHN BRAND OF CRYSTALLINE NOVOBIOCIN SODIUM

IN*

PANMYCIN PHOSPHATE PLUS THE ANTIMONILIAL PROTECTION OF NYSTATIN

The logical choice for patients requiring high doses of antibiotics or prolonged antibiotic therapy; for patients with previous monilial complications; for diabetics; patients on corticoids; the pregnant, debilitated, or elderly; and for infants, especially the premature.

THE CHOICE OF A SYSTEMIC ANTIBIOTIC IS A MATTER OF CLINICAL JUDGMENT

1. PANMYCIN PHOSPHATE IN COMMON MIXED INFECTIONS

USUAL DOSAGE: ADULTS: 250 mg. every 6 hours or 500 mg. every 12 hours. CHILDREN: Approximately 8 mg. per pound of body weight daily, in four equally divided doses every 6 hours, or two equally divided doses every 12 hours.

SUPPLIED: CAPSULES: 250 mg. in bottles of 16 and 100; 125 mg. in bottles of 25 and 100.

PANMYCIN KM SYRUP: Each teaspoonful (5 cc.) contains tetracycline equivalent to 125 mg. tetracycline hydrochloride, and potassium metaphosphate, 100 mg., mint flavor, in 2 fluidounce and pint bottles.

2. PANALBA IN POTENTIALLY SERIOUS INFECTIONS

USUAL DOSAGE: ADULTS: 1 or 2 capsules three or four times a day, depending on the type and severity of the infection. CHILDREN: Proportionately less.

SUPPLIED: Each powder-blue-and-brown capsule contains Panmycin (tetracycline) Phosphate complex equivalent to 250 mg. tetracycline hydrochloride, and Albamycin (as novobiocin sodium) 125 mg.; in bottles of 16 and 100.

Also available: **PANALBA KM GRANULES** (Pediatric). When reconstituted, each 5 cc. teaspoonful contains Panmycin equivalent to tetracycline hydrochloride, 125 mg. and Albamycin (as novobiocin calcium) 62.5 mg., and potassium metaphosphate 100 mg.; in pleasantly flavored vehicle. Dosage is based upon amount of tetracycline—6 to 8 mg. per pound of body weight per day in 2 to 4 equally divided doses.

3. COMYCIN FOR THE 7 MONILIA-SUSCEPTIBLE TYPES

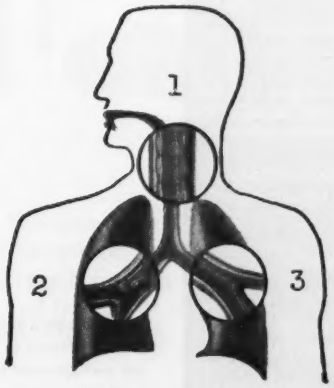
USUAL DOSAGE: ADULTS: 1 or 2 capsules every 6 hours. CHILDREN: Proportionately less.

SUPPLIED: Each brown-and-pink capsule contains tetracycline phosphate complex, equivalent to 250 mg. tetracycline hydrochloride; nystatin 250,000 units. In bottles of 16 and 100.

Upjohn

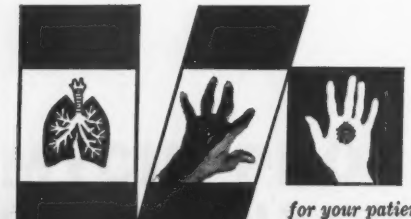
The Upjohn Company, Kalamazoo, Michigan

breaks up cough*



* **Drawing shows how 3-pronged attack of** Pyribenzamine Expectorant with Ephedrine **breaks up cough by: (1) reducing histamine-induced congestion and irritation throughout the respiratory tract; (2) liquefying thick and tenacious mucus; (3) relaxing bronchioles. Pyribenzamine Expectorant with** Codeine **and Ephedrine also available (exempt narcotic). Pyribenzamine® citrate (tripelennamine citrate CIBA).** C I B A

A NEW, CORTICOSTEROID MOLECULE WITH GREATER ANTIALLERGIC, ANTIRHEUMATIC AND ANTI-INFLAMMATORY ACTIVITY



for your patients with

- BRONCHIAL ASTHMA, ALLERGIC DISORDERS
- ARTHRITIC DISORDERS
- DERMATOSES

Squibb Triamcinolone

KENACORT

- far less gastrointestinal distress
- safe to use in asthma with associated cardiac disease; no sodium and water retention
- does not produce secondary hypertension—low salt diet not necessary
- no unnatural psychic stimulation
- often works when other glucocorticoids have failed
- and on a lower daily dosage range

Initial dosage: 8 to 20 mg. daily. After 2 to 7 days gradually reduce to maintenance levels.
See package insert for specific dosages and precautions.
1 mg. tablets, bottles of 50 and 500.
4 mg. tablets, bottles of 30 and 100.

Squibb Quality—the Priceless Ingredient

"KENACORT" IS A SQUIBB TRADEMARK

the chill

the cough

the aching muscles

the fever

Viral upper respiratory infection. . . . For this patient, your management will be twofold—prompt symptomatic relief plus the prevention and treatment of bacterial complications. PEN·VEE·*Cidin* backs your attack by broad, multiple action. It relieves aches and pains, and reduces fever. It counters depression and fatigue. It alleviates cough. It calms the emotional unrest. And it dependably combats bacterial invasion because it is the only preparation of its kind to contain penicillin V.

SUPPLIED: Capsules, bottles of 36. Each capsule contains 62.5 mg. (100,000 units) of penicillin V, 194 mg. of salicylamide, 6.25 mg. of promethazine hydrochloride, 130 mg. of phenacetin, and 3 mg. of mephentermine sulfate.

This advertisement conforms to the Code for Advertising of the Physicians' Council for Information on Child Health.

PEN·VEE·*Cidin*®

Penicillin V with Salicylamide, Promethazine Hydrochloride, Phenacetin, and Mephentermine Sulfate, Wyeth

Wyeth

Philadelphia 1, Pa.

Therapeutic Nutrition in Chronic Disease

Meat...

and Protein Nutrition in Vascular Disease

Whether the eventual solution of the problem of atherogenesis will come out of the field of dietetics, biophysics, or pharmacology, one fact remains undeniable:

Adequate protein nutrition is considered of importance for the age group most commonly affected by disease of the vascular system, so that the demands of good nutritional health might be met.

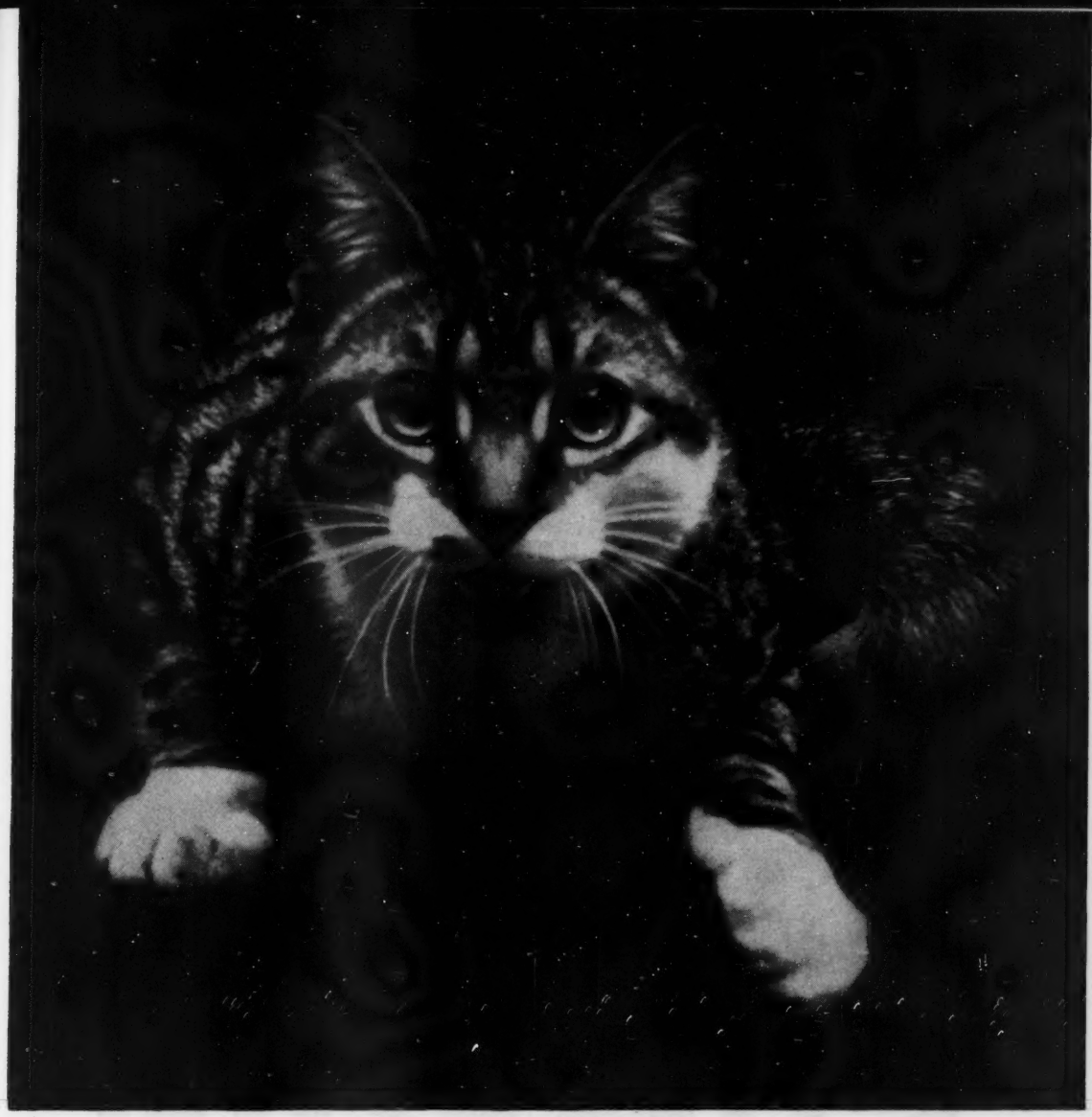
Meat is outstanding among protein foods. It supplies all the essential amino acids, and closely approaches the quantitative proportions needed for biosynthesis of human tissue.

In addition, it is an excellent source of B vitamins, including B_6 and B_{12}, as well as iron, phosphorus, potassium, and magnesium.

When curtailment of fat intake is deemed indicated, meat need not always be denied the patient. Visible fat obviously should not be eaten. But the contained percentage of invisible (interstitial) fat is well within the limits of reasonable fat allowance.

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

American Meat Institute
Main Office, Chicago...Members Throughout the United States



TAKE A NEW LOOK AT ALLERGENS*
TAKE A LOOK AT NEW DĪMETANE

There is no antihistamine better than DIMETANE for allergic protection. DIMETANE gives you good reasons to re-examine the antihistamine you are now using: unexcelled potency, unsurpassed therapeutic index and relative safety...minimum drowsiness or other side effects. Has been effective where other antihistamines have failed. DIMETANE Extentabs® (12 mg.) protect for 10-12 hours on one tablet. Also available: Tablets (4 mg.), Elixir (2 mg. per 5 cc.).

A. H. ROBINS CO., INC., Richmond 20, Virginia
Ethical Pharmaceuticals of Merit Since 1878

*Typical Allergens: Animal Hair and Dander • Pollen • Molds • Bacteria and Viruses • Feathers • Insect Scales • Vegetable Fibers and Seeds Plant Juices • House Dust • Drugs and Chemicals • Minerals and Metals.

Robins

Dīmetane® (PARABROMDYLAMINE MALEATE)

when you encounter

- respiratory infections
- gastrointestinal infections
- genitourinary infections
- miscellaneous infections

for all tetracycline-amenable infections, prescribe superior

SUMYCIN

Squibb Tetracycline Phosphate Complex

In your patients, **SUMYCIN** *produces:*

1. Superior initial tetracycline blood levels—faster and higher than ever before—assuring fast transport of adequate tetracycline to the site of the infection.

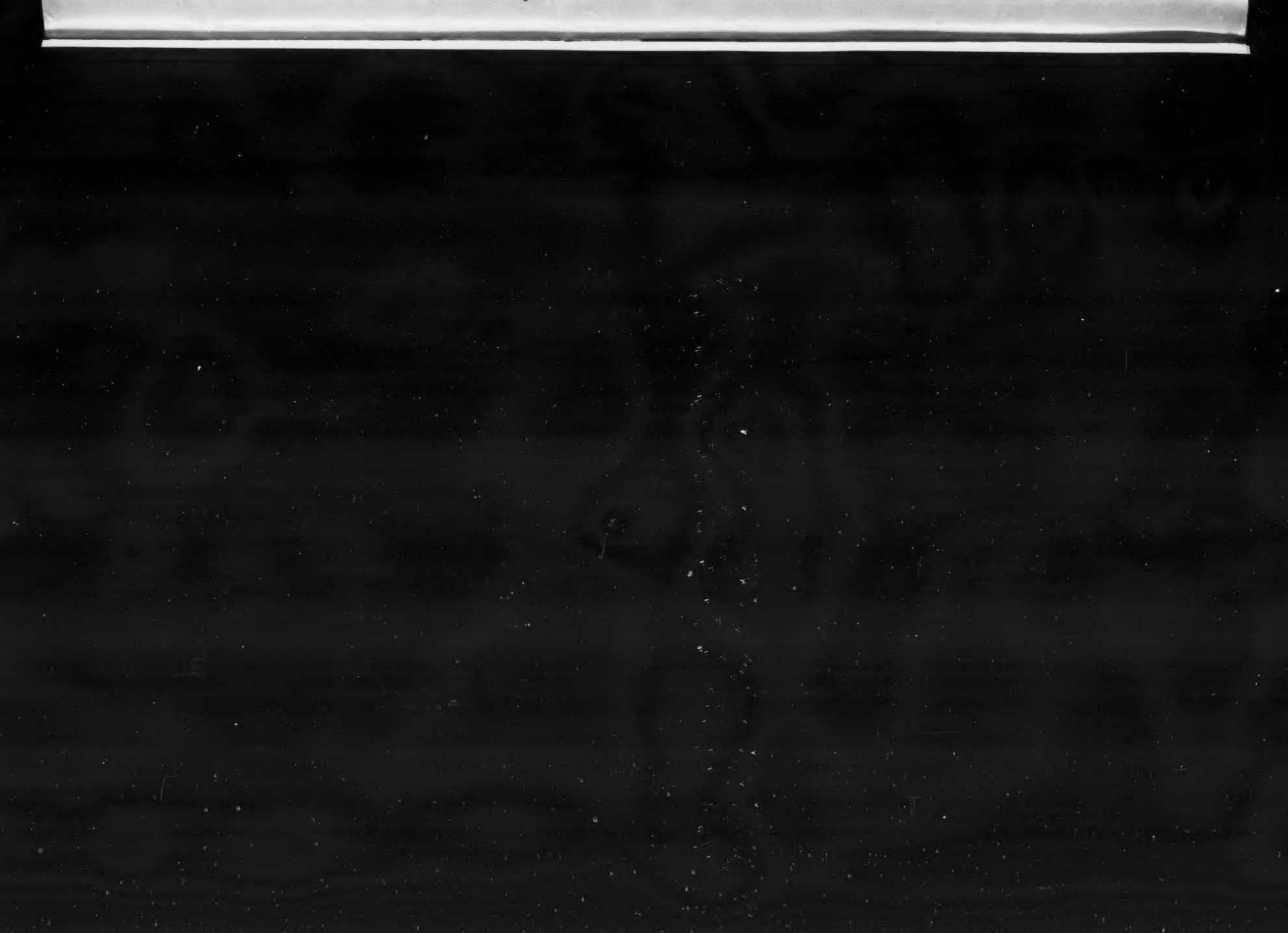
2. High degree of freedom from annoying or therapy-interrupting side effects.

Supply:	*Tetracycline phosphate complex equiv. to tetracycline HCl (mg.)*	*Packaging:*
Sumycin Capsules (per Capsule)	250	Bottles of 16 and 100
Sumycin Suspension (per 5 cc.)	125	2 oz. bottles
Sumycin Pediatric Drops (per cc.—20 drops)	100	10 cc. dropper bottles

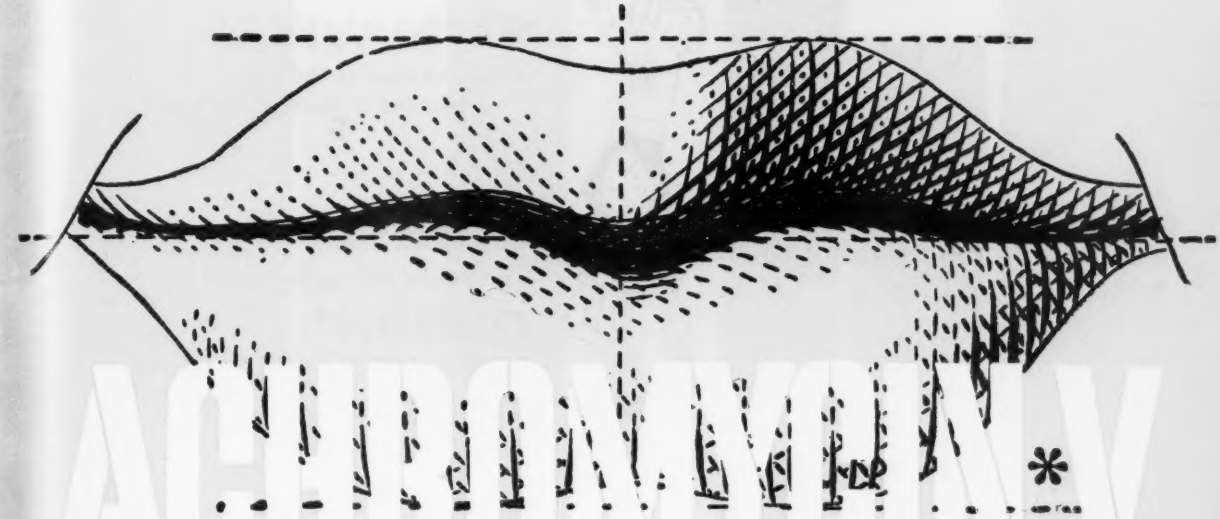
"SUMYCIN" IS A SQUIBB TRADEMARK

SQUIBB

Squibb Quality—the Priceless Ingredient



TASTY, FAST-ACTING ORAL FORM OF CITRATE-BUFFERED ACHROMYCIN V



TETRACYCLINE BUFFERED WITH SODIUM CITRATE

ACHROMYCIN V SYRUP

aqueous

ready-to-use

freely miscible

- accelerated absorption in the gastro-intestinal tract
- early, high peaks of concentration in body tissue and fluid
- quick control of a wide variety of infections
- unsurpassed, true broad-spectrum action
- minimal side effects
- well-tolerated by patients of all ages

ACHROMYCIN V SYRUP:
Orange Flavor. Each teaspoonful (5 cc.) contains 125 mg. of tetracycline, HCl equivalent, citrate-buffered. Bottles of 2 and 16 fl. oz.

DOSAGE:
6-7 mg. per lb. of body weight per day.

*Reg. U.S. Pat. Off.

Lederle

LEDERLE LABORATORIES DIVISION
AMERICAN CYANAMID COMPANY
PEARL RIVER, NEW YORK

See anybody here you know, Doctor?

I'm just too much

AMPLUS®
for sound obesity management
dextro-amphetamine plus vitamins and minerals

I'm too little

STIMAVITE®
stimulates appetite and growth
vitamins B_1, B_6, B_{12}, C and L-lysine

I'm simply two

OBRON®
a nutritional buildup for the OB patient

OBRON® HEMATINIC
when anemia complicates pregnancy

And I'm getting brittle

NEOBON®
5-factor geriatric formula
hormonal, hematinic and nutritional support

ROETINIC®
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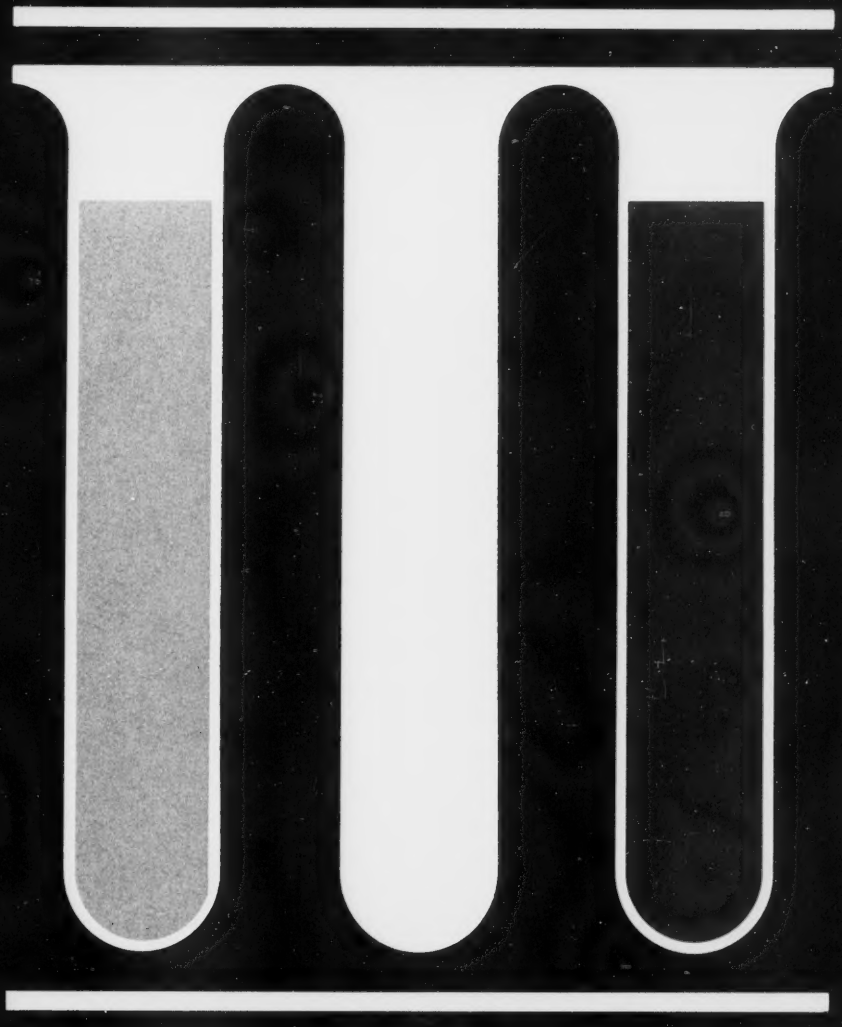

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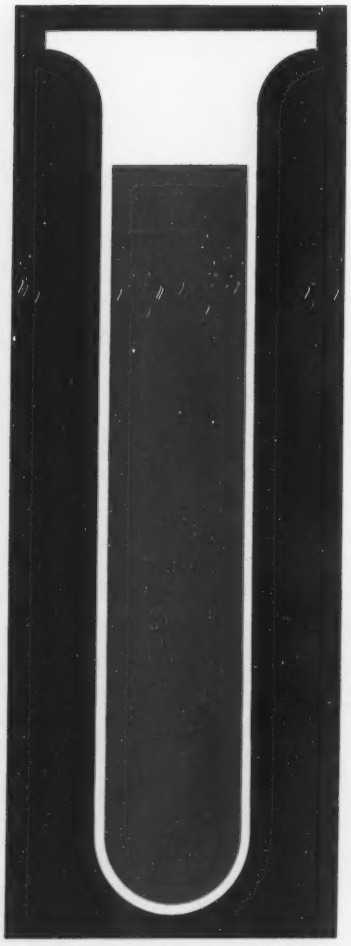
FILMTAB®

STEARATE (Erythromycin Stearate, Abbott)

against staph-, strep- and pneumococci



Indications

ERYTHROCIN is indicated in treating infections caused by staphylococci, streptococci (including enterococci), and pneumococci. Indicated also, in treating infections that have become resistant to other antibiotics. May be used for patients who are allergic to penicillin or other antibacterials.

Dosage

Usually administered in a total daily dose of 1 to 2 Gm., depending on severity of infection. Suggested dose is 250 mg. every six hours; for severe infections, usual dose is 500 mg. every six hours.

Supplied

In bottles of 25 and 100 *Filmtabs* (100 and 250 mg.). Also, in tasty, cinnamon-flavored oral suspension, in 75-cc. bottles. Each 5-cc. teaspoonful represents 100 mg. of ERYTHROCIN activity.

*Filmtab—Film-sealed tablets, Abbott; pat. applied for.

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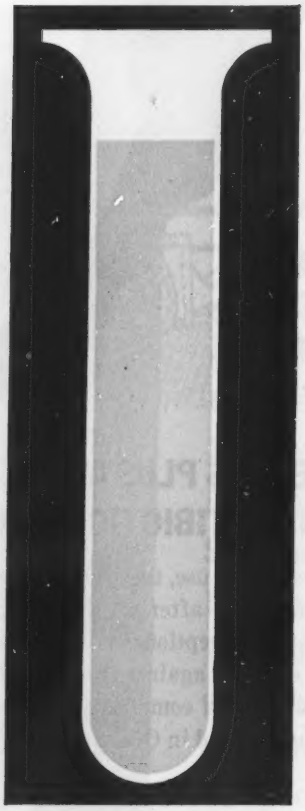
You'll find ERYTHROCIN is highly effective against the majority of coccal infections and may also be used to counteract complications from severe viral attacks. It comes in *Filmtabs* and in Oral Suspension. Abbott

802069

Compocillin-V®

for those penicillin-sensitive organisms



Indications

Against all penicillin-sensitive organisms. For prophylaxis and treatment of complications in viral conditions. And as a prophylaxis in rheumatic fever and in rheumatic heart disease.

Dosage

Depending on the severity of the infection, 125 to 250 mg. (200,000 to 400,000 units) every four to six hours. For children, dosage is determined by age and weight.

Supplied

Filmtabs COMPOCILLIN-V (Potassium Penicillin V, Abbott) come in 125 mg. (200,000 units), bottles of 50; and in 250 mg. (400,000 units), bottles of 25. Oral Suspension COMPOCILLIN-V (Hydrabamine Penicillin V, Abbott), contains 180 mg. per 5-cc. teaspoonful, in 40-cc. and 80-cc. bottles.

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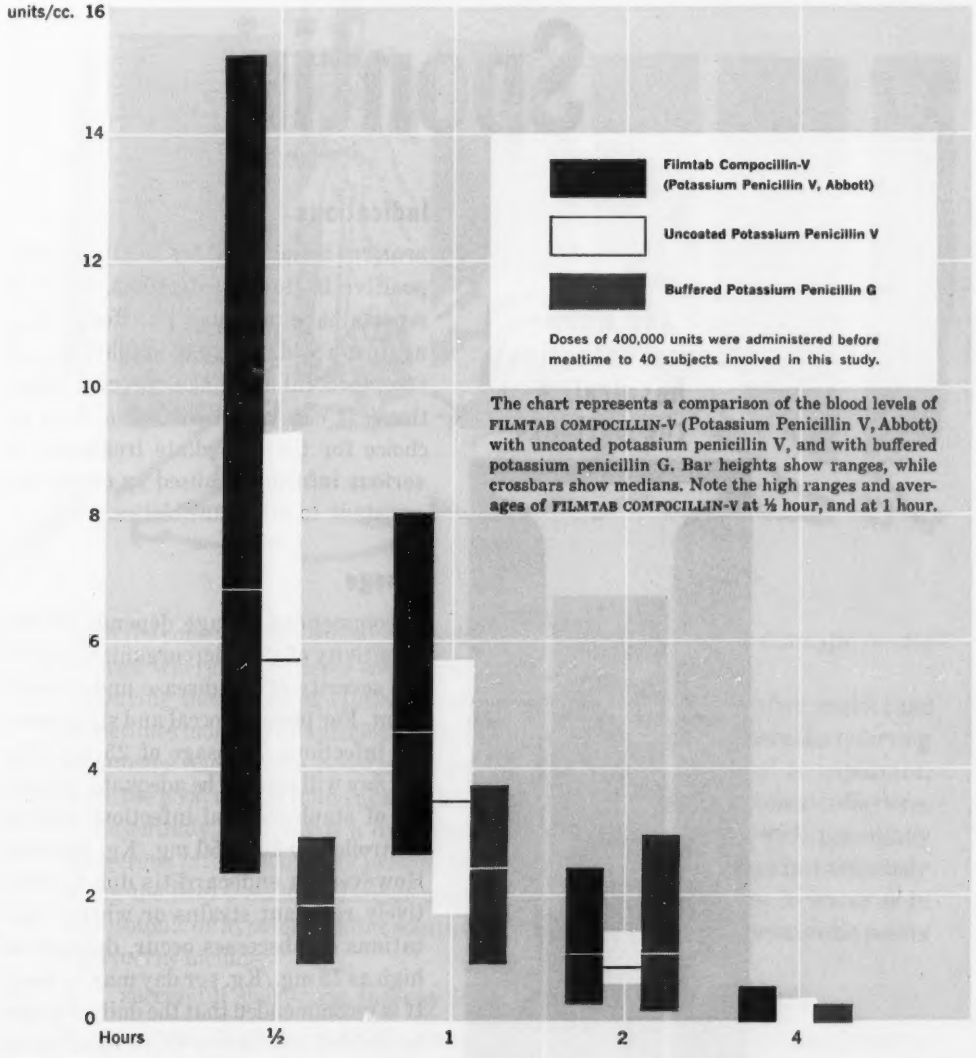

The chart represents a comparison of the blood levels of FILMTAB COMPOCILLIN-V (Potassium Penicillin V, Abbott) with uncoated potassium penicillin V, and with buffered potassium penicillin G. Bar heights show ranges, while crossbars show medians. Note the high ranges and averages of FILMTAB COMPOCILLIN-V at ½ hour, and at 1 hour.

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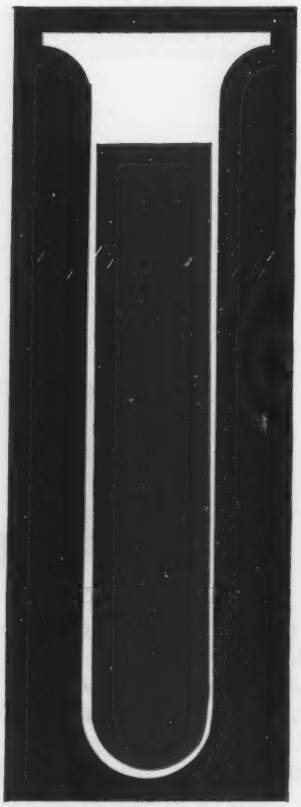
Spontin®

(Ristocetin, Abbott)

and when coccal infections hospitalize the patient



Indications

SPONTIN is indicated for treating gram-positive bacterial infections. Clinical reports have indicated its effectiveness against a wide range of staphylococcal, streptococcal and pneumococcal infections. It can be considered a drug of choice for the immediate treatment of serious infections caused by organisms resistant to other antibiotics.

Dosage

Recommended dosage depends on the sensitivity of the microorganism and on the severity of the disease under treatment. For pneumococcal and streptococcal infections, a dosage of 25 mg./Kg. per day will usually be adequate. Majority of staphylococcal infections will be controlled by 25 to 50 mg./Kg. per day. However, in endocarditis due to relatively resistant strains or where vegetations or abscesses occur, dosages as high as 75 mg./Kg. per day may be used. It is recommended that the daily dosages be divided into two or three equal parts at eight- or twelve-hour intervals.

Supplied

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* J.A.M.A. *166*:158,1958; Welsh, A.L. and Ede, M. "...*prompt remissions of...acute phases.*" with TARCORTIN

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* 1. Clyman, S. G.: Postgrad. Med. *21*:309, 1957.
2. Bleiberg, J.: J. M. Soc. New Jersey *53*:37, 1956.
3. Abrams, B. P. and Shaw, C.: Clin. Med. *3*:839, 1956.
4. Welsh, A. L., and Ede, M.: Ohio State M. J. *50*:837, 1954.
5. Bleiberg, J.: Am. Practitioner *8*:1404, 1957.

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when anxiety and tension "erupts" in the G. I. tract...

IN GASTRIC ULCER

PATHIBAMATE*

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Combines Meprobamate (*400 mg.*) the most widely prescribed tranquilizer . . . helps control the "emotional overlay" of gastric ulcer — without fear of barbiturate loginess, hangover or habituation . . . *with* PATHILON (*25 mg.*) the anticholinergic noted for its extremely low toxicity and high effectiveness in the treatment of many G.I. disorders.

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With type as well as amount of fat in the human diet now assuming such importance, the new role of corn oil as a source of unsaturated fatty acids has prompted these questions:

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3 What is the best way to provide unsaturated fatty acids?

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4 How is corn oil most easily taken in the usual daily diet?

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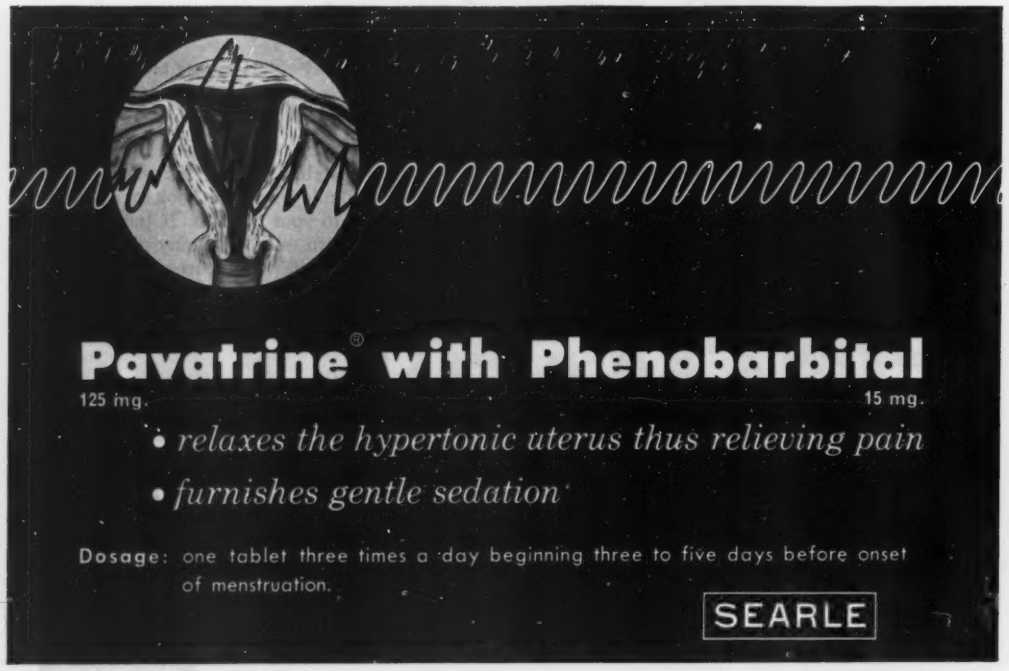
in dysmenorrhea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when anxiety and tension "erupts" in the G. I. tract...

IN DUODENAL ULCER

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Reference: 1. J.A.M.A. 158:386 (June 4) 1955.

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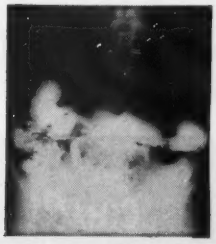
when anxiety and tension "erupts" in the G. I. tract...

IN ILEITIS



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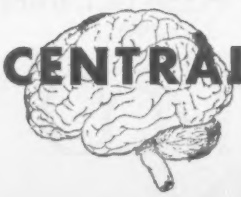

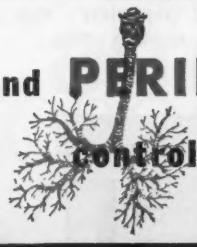

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when anxiety and tension "erupts" in the G. I. tract...

in spastic and irritable colon

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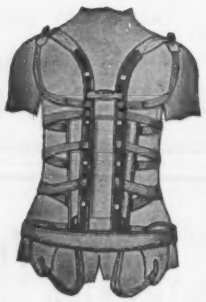

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Index to Advertisers

Abbott Laboratories 39, 40, 53, 54, 55, 56, 57, and facing 52
American Meat Institute 48
Ames Company, Inc. ..... Inside back cover
Ayerst Laboratories 63

Bayer Company 36
Bristol Laboratories, Inc. ..... 14 and 15
Bristol-Myers Company 62
Burroughs Wellcome and Company, Inc. ..... 22, 38, and facing 24

Ciba Pharmaceutical Products, Inc. 45
Corn Products Sales Company ..... 34 and 59

Endo Laboratories 35

Goetze Niemer Company 64

Hanicke, P. W., Manufacturing Company 66
Hotel Commodore 62

Kansas Oxygen, Inc. ..... Center section

Lakeside Laboratories 5
Lattimore-Fink Laboratories 66
Lederle Laboratories ..... 6, 7, 19, 20, 21, 24, 30, center section, 51, 58, 61, 63, and 65
Lilly, Eli, and Company 32

Manx Hotel 60
Medical Protective Company 40
Menninger Foundation 64
Merck, Sharp and Dohme, Inc. ..... 9, 12, 13, 16, 17, 28, 29, and 44
Munns Medical Supply Company, Inc. 60

Neurological Hospital 65

Parke, Davis and Company ..... Inside front cover and 3
Petro's Surgical Appliances 66
Pfizer Laboratories 23
Physicians Casualty Association 40
Prairie View Hospital 65

Quincy X-Ray and Radium Laboratories 65

Reed and Carnrick 58
Robins, A. H., Company, Inc. ..... 10, 11, and 49
Roerig, J. B., and Company, Inc. ..... 18, 25, 37, and 52

Schering Corporation ..... 31, 42, and 43
Searle, G. D., and Company ..... 27, 33, and 60
Seven-Up Bottling Company, Inc. ..... Center section
Smith-Dorsey Company 41
Smith, Kline and French Laboratories ..... Back cover
Southwest Scientific Corporation 65
Squibb, E. R., and Sons ..... 26, 46, and 50

University of Kansas School of Medicine .... Center section
Upjohn Company ..... Facing 44

Wallace Laboratories ..... Facing 8
Winthrop Laboratories ..... 8, 61, and 64
Wyeth Laboratories 47

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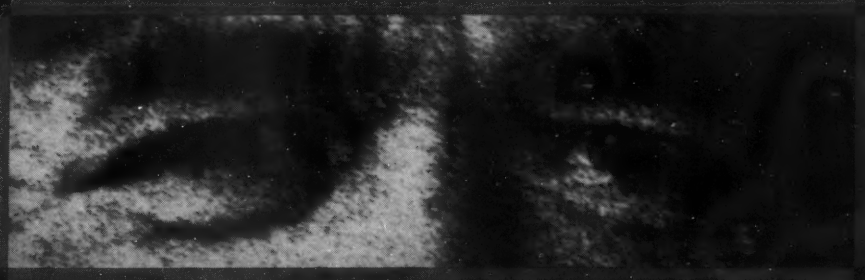

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(1) Don't argue—let patient "talk out" his troubles. (2) Don't counsel—help him solve his own problems. (3) Don't be hostile—allow patient to express hostility without reciprocating. (4) Don't be unsure—stress significance of normal or abnormal physical findings in relation to symptoms. (5) Don't be too reassuring—overoptimism may suggest you take the symptoms too lightly. (6) Don't approve or censure. (7) Don't be too credulous—patients' words may conceal hidden meanings.

Source—Hyman, M.: Some Aspects of Psychiatry in General Practice, GP *16*:83 (Oct.) 1957.

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*Bauer, H. G.; Seegers, W.; Krawzoff, M., and McGavack, T. H.: A Clinical Evaluation of Ectylurea (NOSTYN®), in press.

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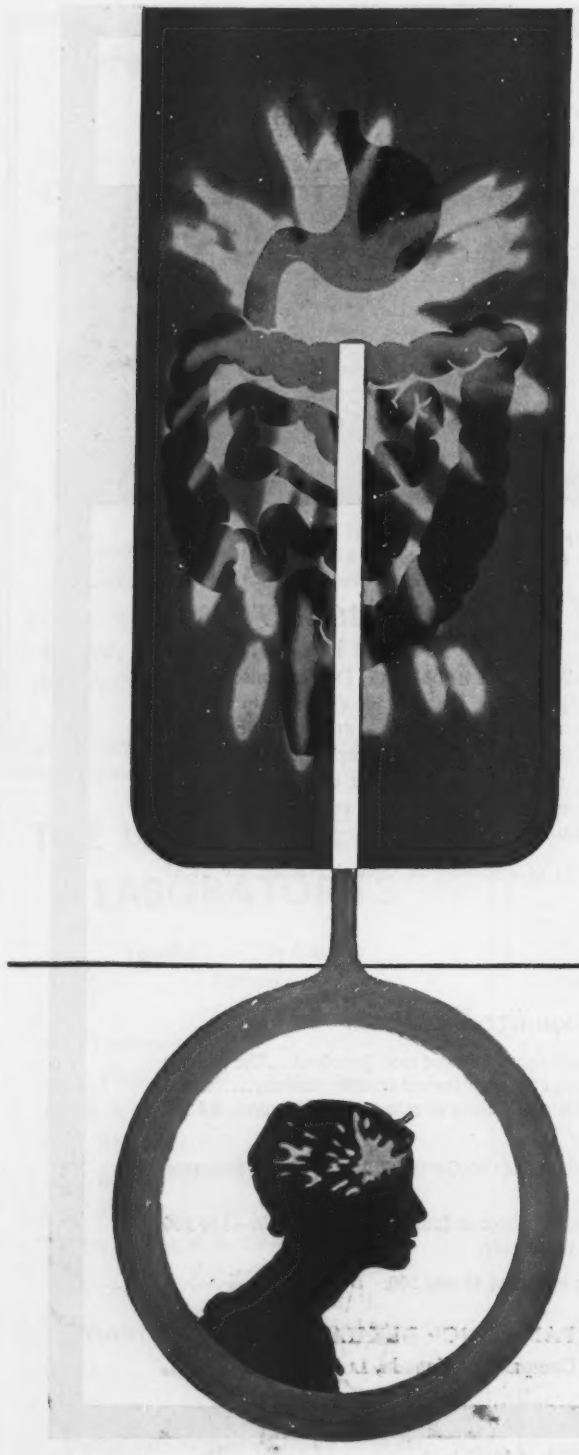

in G.I. disorders

'Compazine' controls tension —often brings complete relief

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